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3544. *The same.* la. 8°. *New York,* 1892.

2nd printing. Interleaved copy, bd. in 2 vols., with numerous MS. notes, and insertions. The latter include a prospectus (endorsed: "This, thank the Lord, I did not write.") and 2 photos. of the author at work on the book. The following are among the notes:

PRIVATE COPY. May all the curses of the good Bishop Ernulphus light on the borrower-and-not-returner or upon the stealer of this book.

The 1st printing (1st copy received Feb. 24th, 1892) of 3,000 copies was distributed by March 17th, on which date I had notice from Appleton & Co. to send corrections for a 2nd printing. This copy—2nd printing—was received April 16th, 1892. For samples of a good and a bad page of the MS. see at the end of this [1st] volume.

THE WRITING OF A TEXT-BOOK.

On several occasions, in Philadelphia, I was asked by Lea Bros. to prepare a work on Diagnosis and had half promised one; indeed I had prepared a couple of chapters, but continually procrastinated on the plea that up to the 40th year a man was fit for better things than text-books. Time went on and as I crossed this date I began to feel that the energy and persistence necessary for the task were lacking. In Sept. 1890 I returned from a four-months trip in Europe, shook myself, and towards the end of the month began a work on Practice. I had nearly finished the chapter on Typhoid Fever when Dr. Granger, Messrs. Appleton's agent, came from N.Y. to ask me to prepare a Text-book on Medicine. We haggled for a few weeks about terms and finally, selling my brains to the Devil, I signed the contract. My intention had been to publish the work myself and have Lippincott or Blakiston (both of whom offered) handle the book, but the bait of a guaranteed circulation of 10,000 copies in two years and fifteen hundred dollars on the date of publication was too glittering, and I was hooked.

October, November, and December were not very satisfactory months and Jan. 1st, 1891, saw the infectious diseases scarcely completed. I then got well into harness. Three mornings of each week I stayed at home and dictated from 8 a.m. till 1 p.m. On the alternate days I dictated after the morning hospital visit, beginning about 11.30. The spare hours of the afternoons were devoted to correction and reference work. Early in May I gave up the house, 209 Monument St., and went to my rooms at the Hospital. The routine there was: 8 a.m. to 1 p.m. dictation, 2 p.m. visit to the private patients and special cases in the wards, after which revision, &c. After 5 p.m. I saw any outside cases; dined at the Club about 6.30, loafed until 9.30, bed at 10, up at 7 a.m. I had arranged to send MS. by July 1st, and on that date I forwarded five sections, but the publishers did not begin to print until the middle of August.

The first two weeks of August I spent in Toronto, and then with the same routine I practically finished the MS. by about October 15th. During the summer the entire MS. was carefully revised for the press by Mr. Powell of the English Department of the University. The last three months of '91 were devoted to proof reading. In January I made out the index, and in the entire work nothing so wearied me as the verifying of every reference. Without the help of Lafleur and Thayer who took the wards off my hands I never could have finished in so short a time; my other assistants also rendered much aid in looking up references and special points.

During the writing of the work I lost only one afternoon through transient indisposition and never a night's rest. Between September 1890 and January 1892 I gained nearly 8 lbs. in weight.

[W. O.]

Relation to nasal obstruction

II. Hypertrophic Emphysema.

This form, also known as substantive or idiopathic emphysema, is a well-marked clinical affection, characterized by enlargement of the lungs, due to distention of the air-cells and atrophy of their walls, and clinically by imperfect aëration of the blood and more or less marked dyspnœa.

Etiology.—Emphysema is the result of persistently high intra-alveolar tension acting upon a congenitally weak lung tissue. If the mechanical views which have prevailed so long as to its origin were true, the disease would certainly be much more common; since violent respiratory efforts, believed to be the essential factor, are performed by a majority of the working classes. Strongly in favor of the view that the nutritive change in the air-cells is the primary factor is the markedly hereditary character of the disease and the frequency with which it starts early in life. These are two points upon which scarcely sufficient stress has been laid. To James Jackson, Jr., of Boston, we owe the first observations on the hereditary character of emphysema. Working under Louis's directions, he found that in 18 out of 28 cases one or both parents were affected.

I have been impressed by the frequency of the condition in children, and the number of cases in which on inquiry symptoms pointing to the occurrence of the disease in childhood can be obtained. It may develop, too, in several members of the same family. We are still ignorant as to the nature of this congenital pulmonary weakness. Cohnheim thinks it probably due to a defect in the development of the elastic-tissue fibres, a statement which is borne out by Eppinger's observations.

Heightened pressure within the air-cells may be due to forcible inspiration or expiration. Much discussion has taken place as to the part played by these two acts in the production of the disease. The inspiratory theory was advanced by Laennec and subsequently modified by Gairdner, who held that in the chronic bronchitis areas of collapse were induced, and compensatory distention took place in the adjacent lobules. This unquestionably does occur in the vicarious or compensatory emphysema, but it probably is not a factor of much moment in the form now under consideration. The expiratory theory, which was supported by Mendelsohn and Jenner, accounts for the condition in a much more satisfactory way. In all straining efforts and violent attacks of coughing, the glottis is closed and the chest walls are strongly compressed by muscular efforts, so that the strain is thrown upon those parts of the lung least protected, as the apices and the anterior margins, in which we always find the emphysema most advanced. The sternum and costal cartilages gradually yield to the heightened intrathoracic pressure and are, in advanced cases, pushed forward, giving the characteristic rotundity to the thorax. As mentioned, the cartilages gradually become calcified. One theory of

the disease is that there is a gradual enlargement of the thorax and the lungs increase in volume to fill up the space.

Of other etiological factors occupation is the most important. The disease is met with in players on wind instruments, in glass-blowers, and in occupations necessitating heavy lifting or straining. Whooping-cough and bronchitis play an important *rôle*, not so much in the changes which they induce in the bronchi as in consequence of the prolonged attacks of coughing.

Morbid Anatomy.—The thorax is capacious, usually barrel-shaped, and the cartilages are calcified. On removal of the sternum, the anterior mediastinum is found completely occupied by the edges of the lungs, and the pericardial sac may not be visible. The organs are very large and have lost their elasticity, so that they do not collapse either in the thorax or when placed on the table. The pleura is pale and there is often an absence of pigment, sometimes in patches, termed by Virchow *albinism* of the lung. To the touch they have a peculiar, downy, feathery feel, and pit readily on pressure. This is one of the most marked features. Beneath the pleura greatly enlarged air-vesicles may be readily seen. They vary in size from ½ to 3 mm., and irregular bullæ, the size of a walnut or larger, may project from the free margins. The best idea of the extreme rarefaction of the tissue is obtained from sections of a lung distended and dried. At the anterior margins the structure may form an irregular series of air-chambers, resembling the frog's lung. On careful inspection with the hand-lens, remnants of the interlobular septa or even of the alveoli may be seen on these large emphysematous vesicles. Though general throughout the organs, the distention is more marked, as a rule, at the anterior margins, and is often specially developed at the inner surface of the lobe near the root, where in extreme cases air-spaces as large as an egg may sometimes be found. Microscopically there is seen atrophy of the alveolar walls, by which is produced the coalescence of neighboring air-cells. In this process the capillary network disappears before the walls are completely atrophied. The loss of the elastic tissue is a special feature. It is stated, indeed, that in certain cases there is a congenital defect in the development of this tissue. The epithelium of the air-cells undergoes a fatty change, but the large distended air-spaces retain a pavement layer.

The bronchi in emphysema show important changes. In the larger tubes the mucous membrane may be rough and thickened from chronic bronchitis; often the longitudinal lines of submucous elastic tissue stand out prominently. In the advanced cases many of the smaller tubes are dilated, particularly when, in addition to emphysema, there are peribronchial fibroid changes. Bronchiectasis is not, however, an invariable accompaniment of emphysema, but, as Laennec remarks, it is difficult to understand why it is not more common. Of associated morbid changes the most important are found in the heart. The right chambers are

dilated and hypertrophied, the tricuspid orifice is large, and the valve segments are often thickened at the edges. In advanced cases the cardiac hypertrophy is general. The pulmonary artery and its branches may be wide and show marked atheromatous changes.

The changes in the other organs are those commonly associated with prolonged venous congestion.

Symptoms.—The disease may be tolerably advanced before any special symptoms develop. A child, for instance, may be somewhat short of breath on going up-stairs or may be unable to run and play as other children without great discomfort; or, perhaps, has attacks of slight lividity. Doubtless much depends upon the completeness of cardiac compensation. When this is perfect, there may be no special interruption of the pulmonary circulation and, except in violent exertion, there is no interference with the aëration of the blood. In well-developed cases the following are the most important symptoms: *Dyspnœa*, which may be felt only on slight exertion, or may be persistent, and aggravated by intercurrent attacks of bronchitis. The respirations are often harsh and wheezy, and expiration is distinctly prolonged.

Cyanosis of an extreme grade is more common in emphysema than in other affections with the exception of congenital heart-disease. So far as I know it is the only disease in which a patient may be able to go about and even to walk into the hospital or consulting-room with a lividity of startling intensity. The contrast between the extreme cyanosis and the comparative comfort of the patient is very striking. In other affections of the heart and lungs associated with a similar degree of cyanosis the patient is invariably in bed and usually in a state of orthopnœa.

Bronchitis with associated cough is a frequent symptom and often the direct cause of the pulmonary distress. The contrast between emphysematous patients in the winter and summer is marked in this respect. In the latter they may be comfortable and able to attend to their work, but with the cold and changeable weather they are laid up with attacks of bronchitis. Finally, in fact, the two conditions become inseparable and the patient has persistently more or less cough. The acute bronchitis may produce attacks not unlike asthma. In some instances this is true spasmodic asthma, with which emphysema is frequently associated.

As age advances and with successive attacks of bronchitis the condition gets slowly worse. In hospital practice it is common to admit patients over sixty with well-marked signs of advanced emphysema. The affection can generally be told at a glance—the rounded shoulders, barrel chest, the thin yet oftentimes muscular form, and sometimes, I think, a very characteristic facial expression.

There is another group, however, of younger patients from twenty-five to forty years of age who winter after winter have attacks of intense cyanosis in consequence of an aggravated bronchial catarrh. On inquiry we find that these patients have been short-breathed from infancy, and they

belong, I believe, to a category in which there has been a primary defect of structure in the lung tissue.

Physical Signs.—*Inspection.*—The thorax is markedly altered in shape; the antero-posterior diameter is increased and may be even greater than the lateral, so that the chest is barrel-shaped. The appearance is somewhat as if the chest was in a permanent inspiratory position. The sternum and costal cartilages are prominent. The lower zone of the thorax looks large and the intercostal spaces are much widened, particularly in the hypochondriac regions. The sternal fossa is deep, the clavicles stand out with great prominence, and the neck looks shortened from the elevation of the thorax and the sternum. A zone of dilated venules may be seen along the line of attachment of the diaphragm. Though this is common in emphysema, it is by no means peculiar to it. Andrew, of Bartholomew's Hospital, and, according to Duckworth, Laycock have called attention to it. This network in the lower thoracic region, just above the costal margin and following its curves, is a well-marked feature in many persons, and is seen not only in emphysema, but in many cases of hepatic trouble.

Behind, the curve of the spine is increased and the back is remarkably rounded, so that the scapulæ seem to be almost horizontal. ~~Mensuration shows the rounded form of the chest; the antero-posterior diameter may exceed the transverse~~. The respiratory movements, which may look energetic and forcible, exercise little or no influence. The chest does not expand, but there is a general elevation. The inspiratory effort is short and quick; the expiratory movement is prolonged. There may be retraction instead of distention in the upper abdominal region during inspiration, and there is sometimes seen a transverse curve crossing the abdomen at the level of the twelfth rib. The apex beat of the heart is not visible, and there is usually marked pulsation in the epigastric region. The cervical veins stand out prominently and may pulsate.

Palpation.—The vocal fremitus is somewhat enfeebled but not lost. The apex beat can rarely be felt. There is a marked shock in the lower sternal region and very distinct pulsation in the epigastrium. *Percussion* gives greatly increased resonance, full and drum-like—what is sometimes called hyperresonance. The note is not often distinctly tympanitic in quality. The percussion note is greatly extended, the heart dulness may be obliterated, the upper limit of liver dulness is greatly lowered, and the resonance may extend to the costal margin. Behind, a clear percussion note extends to a much lower level than normal. The level of splenic dulness, too, may be lowered.

On *auscultation* the breath-sounds are usually enfeebled and may be masked by bronchitic râles. The most characteristic feature is the prolongation of the expiration, and the normal ratio may be reversed—4 to 1 instead of 1 to 4. It is often wheezy and harsh and associated with coarse râles and sibilant rhonchi. It is said that in interstitial emphysema there

may be a friction sound heard not unlike that of pleurisy. As already noted, the cardiac impulse may be barely felt in the lower sternal region. The heart-sounds are usually clear; but in advanced cases, when there is marked cyanosis, a tricuspid regurgitant murmur may be heard. Accentuation of the pulmonary second sound is present.

The *course* of the disease is slow but progressive, the recurring attacks of bronchitis aggravating the condition. Death may occur from intercurrent pneumonia, either lobar or lobular, and dropsy may supervene from cardiac failure. Occasionally death results from overdistention of the heart, with extreme cyanosis. Duckworth has called attention to fatal hæmorrhage in emphysema. It certainly is not common. In an old emphysematous patient at the Montreal General Hospital death followed the erosion of a main branch of the pulmonary artery by an ulcer near the bifurcation of the trachea.

Treatment.—Practically, the measures mentioned in connection with bronchitis should be employed. No remedy is known which has any influence over the progress of the condition itself. Bronchitis is the great danger of these patients, and therefore when possible they should live in an equable climate. In consequence of the venous engorgement they are liable to gastric and intestinal disturbance, and it is particularly important to keep the bowels regulated and to avoid the flatulency which often seriously aggravates the dyspnœa. Patients who come into the hospital in a state of urgent dyspnœa and lividity, with great engorgement of the veins, particularly if they are young and vigorous, should be bled freely. On more than one occasion I have saved the lives of persons in this condition by venesection. Inhalation of oxygen may be used and the remedies given already mentioned in connection with bronchitis. Strychnine will be found specially useful.

III. Atrophic Emphysema.

This is really a senile change and is called by Sir William Jenner small-lunged emphysema. It is really a primary atrophy of the lung, coming on in advanced life, and scarcely constitutes a special affection. It occurs in "withered-looking old persons" who may perhaps have had a winter cough and shortness of breath for years. In striking contrast to the essential or hypertrophic emphysema, the chest in this form is small. The ribs are obliquely placed, the decrease in the diameter being due to greatly increased obliquity in the position of the ribs. The thoracic muscles are usually atrophied. In advanced cases of this affection the lung presents a remarkable appearance, being converted into a series of large vesicles, on the walls of which the remnants of air-cells may be seen. It is a condition for which nothing can be done.

VI. GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is not an affection *per se*, but occurs in a variety of conditions when necrotic areas undergo putrefaction. It is not easy to say why sphacelus should occur in one case and not in another, as the germs of putrefaction are always in the air-passages, and yet necrotic territories rarely become gangrenous. Total obstruction of a pulmonary artery, as a rule, causes infarction, and the area shut off does not often, though it may, sphacelate. Another factor would seem to be necessary—probably a lowered tissue resistance, the result of general or local causes. It is met with (1) as a sequence of lobar pneumonia. This rarely occurs in a previously healthy person—more commonly in the debilitated or in the diabetic subject. (2) Gangrene is very prone to follow the aspiration pneumonia, since the foreign particles rapidly undergo putrefactive changes. Of a similar nature are the cases of gangrene due to perforation of cancer of the œsophagus into the lung or into a bronchus. (3) The putrid contents of a bronchiectatic, more commonly of a tuberculous, cavity may excite gangrene in the neighboring tissues. The pressure bronchiectasis following aneurism or tumor may lead to extensive sloughing. (4) Gangrene may follow simple embolism of the pulmonary artery. More commonly, however, the embolus is derived from a part which is mortified or comes from a focus of bone disease. Lastly, gangrene of the lung may occur in conditions of debility during convalescence from protracted fever—occasionally, indeed, without our being able to assign any reasonable cause.

Morbid Anatomy.—Laennec, who first accurately described pulmonary gangrene, recognized a diffuse and a circumscribed form. The former, though rare, is sometimes seen in connection with pneumonia, more rarely after obliteration of a large branch of the pulmonary artery. It may involve the greater part of a lobe, and the lung tissue is converted into a horribly offensive greenish-black mass, torn and ragged in the centre. In the circumscribed form there is well-marked limitation between the gangrenous area and the surrounding tissue. The focus may be single or there may be two or more. The lower lobe is more commonly affected than the upper, and the peripheral more than the central portion of the lung. A gangrenous area is at first uniformly greenish brown in color; but softening rapidly takes place with the formation of a cavity with shreddy, irregular walls and a greenish, offensive fluid. The lung tissue in the immediate neighborhood shows a zone of deep congestion, often consolidation, and outside this an intense œdema. In the embolic cases the plugged artery can sometimes be found. When rapidly extending, vessels may be opened and violent hæmorrhage ensue. Perforation of the pleura is not uncommon. The irritating decomposing material usually excites the most intense bronchitis. Embolic processes are not infrequent. There is a remarkable association in some cases between circumscribed

gangrene of the lung and abscess of the brain. I have seen two such cases. One of these, a young man, an Arab, was brought to the University Hospital, almost exsanguine from pulmonary hæmorrhage. He gradually recovered. There were very limited signs in the middle lobe of the right lung, which persisted, but no bacilli were found. There was no fetor of the breath. Weeks afterward he developed severe headache, and in a few days became comatose and died. There was a circumscribed area of healing gangrene at the margin of the lung with great increase of fibrous tissue about it. The artery going to this somewhat wedge-shaped area was obliterated. The contents of the encapsulated cavity were very fetid. There was a large limited abscess in the parieto-temporal region on the right side.

Symptoms and Course.—Usually definite symptoms of local pulmonary disease precede the characteristic features of gangrene. These, of course, are very varied, depending on the nature of the trouble. The sputum is very characteristic. It is intensely fetid—usually profuse—and, if expectorated into a conical glass, separates into three layers—a greenish-brown, heavy sediment; an intervening thin liquid, which sometimes has a greenish or a brownish tint; and, on top, a thick, frothy layer. Spread on a glass plate, the shreddy fragments of lung tissue can readily be picked out. Microscopically, elastic fibres are found in abundance, with granular matter, pigment grains, fatty crystals, bacteria, and leptothrix. It is stated that elastic tissue is sometimes absent, but I have never met with such an instance. The peculiar plugs of sputum which occur in bronchiectasy are not found. Blood is often present, and, as a rule, is much altered. The sputum has, in a majority of the cases, an intensely fetid odor, which is communicated to the breath and may permeate the entire room. It is much more offensive than in fetid bronchitis or in abscess of the lung. The fetor is particularly marked when there is free communication between the gangrenous cavities and the bronchi. On several occasions I have found, post mortem, localized gangrene, which had been unsuspected during life, and in which there had been no fetor of the breath.

The physical signs, when extensive destruction has occurred, are those of cavity, but the limited circumscribed areas may be difficult to detect. Bronchitis is always present.

Among the general symptoms may be mentioned fever, usually of moderate grade; the pulse is rapid, and very often the constitutional depression is severe. But the only special features indicative of gangrene are the sputa and the fetor of the breath. The patient generally sinks from exhaustion. Fatal hæmorrhage may ensue. I have already mentioned a case in which a hæmorrhage from a circumscribed gangrene nearly proved fatal, and I have seen one fatal instance after pneumonia.

Treatment.—The treatment of gangrene is very unsatisfactory. The indications, of course, are to disinfect the gangrenous area, but this is

often impossible. An antiseptic spray of carbolic acid may be employed. A good plan is for the patient to use over the mouth and nose an inhaler, which may be charged with a solution of carbolic acid or creosote. If the signs of cavity are distinct an attempt should be made to cleanse it by direct injections of an antiseptic solution. If the patient's condition is good and the gangrenous region can be localized, an attempt should be made to treat it surgically. Successful cases have been reported. The general condition of the patient is always such as to demand the greatest care in the matter of diet and nursing.

VII. ABSCESS OF THE LUNG.

Etiology.—Suppuration occurs in the lung under the following conditions: (1) As a sequence of inflammation, either lobar or lobular. Apart from the purulent infiltration this is unquestionably rare, and even in lobar pneumonia the abscesses are of small size and usually involve, as Addison remarked, several points at the same time. On the other hand, abscess formation is extremely frequent in the deglutition and aspiration forms of lobular pneumonia. After wounds of the neck or operations upon the throat, in suppurative disease of the nose or larynx, occasionally even of the ear (Volkmann), infective particles reach the bronchial tubes by aspiration and excite an intense inflammation which often ends in suppuration. Cancer of the œsophagus, perforating the root of the lung or into the bronchi, may produce extensive suppuration. The abscesses vary in size from a walnut to an orange, and have ragged and irregular walls, and purulent, sometimes necrotic, contents.

(2) Embolic, so-called metastatic, abscesses, the result of infectious emboli, are extremely common in a large proportion of all cases of pyæmia. They may occur in enormous numbers and present very definite characters. As a rule they are superficial, beneath the pleura, and often wedge-shaped. At first firm, grayish red in color, and surrounded by a zone of intense hyperæmia, suppuration soon follows with the formation of a definite abscess. The pleura is usually covered with greenish lymph, and perforation sometimes takes place with the production of pneumothorax.

(3) Perforation of the lung from without, lodgment of foreign bodies, and, in the right lung, perforation from abscess of the liver or suppurating echinococcus cyst are occasional causes of pulmonary abscess.

(4) Suppurative processes play an important part in chronic pulmonary tuberculosis, many of the symptoms of which are due to them.

Symptoms.—Abscess following pneumonia is easily recognized by an aggravation of the general symptoms and by the physical signs of cavity and the characters of the expectoration. Embolic abscesses cannot often be recognized, and the local symptoms are generally masked in the

general pyæmic manifestations. The characters of the sputum are of great importance in determining the presence of abscess. The odor is offensive, yet it rarely has the horrible fetor of gangrene or of putrid bronchitis. In the pus fragments of lung tissue can be seen, and the elastic tissue may be very abundant. The presence of this with the physical signs rarely leaves any question as to the nature of the trouble. Embolic cases usually run a fatal course. Recovery occasionally occurs after pneumonia.

Medicinal treatment is of little avail in abscess of the lung. When well defined and superficial, an attempt should always be made to open and drain it. A number of successful cases have already been treated in this way.

VIII. PNEUMONOKONIOSIS.

Under this term, introduced by Zenker, is embraced those diseases of the lungs due to the inhalation of dusts in various occupations. They have received various names, according to the nature of the inhaled particles—*anthracosis*, or coal-miner's disease; *siderosis*, due to the inhalation of metallic dusts, particularly iron; *chalicosis*, due to the inhalation of mineral dusts, producing the so-called stone-cutter's phthisis, or the "grinder's rot" of the Sheffield workers.

The dust particles inhaled into the lungs are dealt with extensively by the ciliated epithelium and by the phagocytes, which exist normally in the respiratory organs. The ordinary mucous corpuscles take in a large number of the particles, which fall upon the trachea and main bronchi. The cilia sweep the mucus out to a point from which it can be expelled by coughing. It is doubtful if the particles ever reach the air-cells, but the swollen alveolar cells (in which they are in numbers) probably pick them up on the way. The mucous and the alveolar cells are the normal respiratory scavengers. In dwellers in the country, in which the air is pure, they are able to prevent the access of dust particles to the lung tissue, so that even in adults these organs present a rosy tint, very different from the dark, carbonized appearance of the lungs of dwellers in cities. When the impurities in the air are very abundant, a certain proportion of the dust particles escapes these cells and penetrates the mucosa, reaching the lymph spaces, where they are attacked at once by the cells of the connective-tissue stroma, which are capable of ingesting and retaining a large quantity In coal-miners, coal-heavers, and others whose occupations necessitate the constant breathing of a very dusty atmosphere even these forces are insufficient. Many of the particles enter the lymph stream and, as Arnold has shown in his beautiful researches, are carried (1) to the lymph nodules surrounding the bronchi and blood-vessels; (2) to the interlobular septa beneath the pleura, where they lodge in and between the tissue elements; and (3) along the larger lymph channels to the substernal, bronchial and tracheal glands, in which the stroma cells of

the follicular cords dispose of them permanently and prevent them from entering the general circulation. Occasionally in anthracosis the carbon grains do reach the general circulation, and the coal dust is found in the liver and spleen. As Weigert has shown, this occurs when the densely pigmented bronchial glands closely adhere to the pulmonary veins, through the walls of which the carbon particles pass to the general circulation. The lung tissue has a remarkable tolerance for these particles, probably because a large proportion of them is warehoused, so to speak, in protoplasmic cells. By constant exposure a limit is reached, and there is brought about a very definite pathological condition, an interstitial sclerosis. In coal-miners this may occur in patches, even before the lung tissue is uniformly infiltrated with the dust. In others it appears only after the entire organs have become so laden that they are dark in color, and an ink-like juice flows from the cut surface. The lungs of a miner may be black throughout and yet show no local lesions and be everywhere crepitant.

As already mentioned, the particles are deposited in large numbers in the follicular cords of the tracheal and bronchial glands and of the peri-bronchial and peri-arterial lymph nodules, and in these they finally excite proliferation of the connective-tissue elements. It is by no means uncommon to find in persons whose lungs are only moderately carbonized the bronchial glands sclerosed and hard. In anthracosis the fibroid changes usually begin in the peri-bronchial lymph tissue, and in the early stage of the process the sclerosis may be largely confined to these regions. A Nova Scotian miner, aged thirty-six, died under my care, at the Montreal General Hospital, of black small-pox, after an illness of a few days. In his lungs (externally coal-black) there were round and linear patches ranging in size from a pea to a hazel-nut, of an intensely black color, airless and firm, and surrounded by a crepitant tissue, slate-gray in color. In the centre of each of these areas was a small bronchus. Many of them were situated just beneath the pleura, and formed typical examples of limited fibroid broncho-pneumonia. In addition there is usually thickening of the alveolar walls, particularly in certain areas. By the gradual coalescence of these fibroid patches large portions of the lung may be converted into firm grayish-black, in the case of the coal-miner—steel-gray, in the case of the stone-worker—areas of cirrhosis. In the case of a Cornish miner, aged sixty-three, who died under my care, one of these fibroid areas measured 18 by 6 cm. and 4·5 cm. in depth.

A second important factor in these cases is chronic bronchitis, which is present in a large proportion and really causes the chief symptoms. A third is the occurrence of emphysema, which is almost invariably associated with long-standing cases of pneumonokoniosis. With the changes so far described, unless the cirrhotic area is unusually extensive, the case may present the features of chronic bronchitis with emphysema, but finally another element comes into play. In the fibroid areas softening occurs,

probably a process of necrosis similar to that by which softening is produced in fibro-myomata of the uterus. At first these are small and contain a dark liquid. Charcot calls them, as already mentioned, *ulcères du poumon*. They rarely attain a large size unless a communication is formed with the bronchus, in which case they may become converted into suppurating cavities. The question has been much discussed of late as to what part the tubercle bacillus plays in these cases of pneumonokoniosis with cavity formation. In some instances there is certainly a tuberculous process ingrafted, but that large excavations may occur, or in other instances bronchiectasis without the presence of bacillis, I have convinced myself by the examination of several characteristic specimens.

The *siderosis* induced by the oxide of iron causes an interstitial pneumonia similar to anthracosis. Workers in brass and in bronze are liable to a similar affection.

Chalicosis, due to the deposit of particles of silex and alumina, is found in the makers of mill-stones, particularly the French mill-stones, and also in knife and axe grinders and stone-cutters. Anatomically, this form is characterized by the production of nodules of various sizes, which are cut with the greatest difficulty and sometimes present a curious grayish, even glittering, crystalloid appearance.

Workers in flax and in cotton, and grain-shovellers are also subject to these chronic interstitial changes in the lungs. In all these occupations, as shown by Greenhow, to whose careful studies we owe so much of our knowledge of these diseases, the condition of the lung may ultimately be almost identical.

The *symptoms* do not come on until the patient has worked for a variable number of years in the dusty atmosphere. As a rule there are cough and failing health for a prolonged period of time before complete disability. The coincident emphysema is responsible in great part for the shortness of breath and wheezy condition of these patients. The expectoration is usually muco-purulent, often profuse; in a case of anthracosis, very dark in color—the so-called "black spit"; in a case of chalicosis there may be seen under the microscope the bright angular particles of silica.

Even when there are physical signs of cavity tubercle bacilli are not necessarily, and indeed in my experience they are not usually present. It is remarkable for how long a time a coal-miner may continue to bring up sputum laden with coal particles even when there are only signs of a chronic bronchitis. Many of the particles are contained in the cells of the alveolar epithelium. In these instances it appears that an attempt is made by the leucocytes to rid the lungs of some of the carbon grains.

The *diagnosis* of the condition is rarely difficult; the expectoration is usually characteristic. It must always be borne in mind that chronic

bronchitis and emphysema form essential parts of the process and that in late stages there may be tuberculous infection.

The *treatment* of the condition is practically that of chronic bronchitis and emphysema.

IX. NEW GROWTHS IN THE LUNGS.

Etiology and Morbid Anatomy.—While primary tumors are rare, secondary growths are not uncommon.

The primary growths of the lung are either encephaloid, scirrhus or epithelioma. Recent observations show that the latter is the most common form. Sarcoma also is occasionally found as a primary growth, and still more rarely enchondroma.

The secondary growths may be of various forms. Most commonly they follow tumors in the digestive or genito-urinary organs; not infrequently also tumors of the bone. There may be encephaloid, scirrhus, epithelioma, colloid, melano-sarcoma, enchondroma, or osteoma.

Primary cancer or sarcoma usually involves only one lung. The secondary growths are distributed in both. The primary growth generally forms a large mass, which may occupy the greater part of a lung. Occasionally the secondary growths are solitary and confined chiefly to the pleura, as in a remarkable example which came under my observation, in which the disease was secondary to a myelo-sarcoma of the wrist. The tumor mass occupied a large portion of the left side of the thorax. It grew from the pleura and extended only slightly into the lung, which was compressed and airless. The metastatic growths are nearly always disseminated. Occasionally they occupy a large portion of the pulmonary tissue. In a case of colloid cancer secondary to cancer of the pancreas, I found both lungs voluminous, heavy, only slightly crepitant, and occupied by circular translucent masses, varying in size from a pea to a large walnut.

There are numerous accessory lesions in the pulmonary new growths. There may be pleurisy, either cancerous or sero-fibrinous. The effusion may be hæmorrhagic, but in 200 cases of cancer, primary or secondary, of the lungs and pleura analyzed by Moutard-Martin, hæmorrhagic effusion occurred in only twelve per cent. The tracheal and bronchial glands are usually affected, the cervical glands not infrequently, and occasionally even the inguinal.

The disease is most common in the middle period of life. The primary form affects the sexes equally, but secondary cancer is much more frequent in women than in men. The conditions which predispose to it are quite unknown. It is a remarkable fact that the workers in the Schneeberg cobalt mines are very liable to primary cancer of the lungs. It is stated that in this region a considerable proportion of all deaths in persons over forty are due to this disease.

New growths are occasionally primary in the pleura

(Harris. Jr. M. Coll. Vol. ii, p. 182.

Symptoms.—The clinical features of neoplasms of the lungs are by no means distinctive, particularly in the case of primary growths. The patient may, indeed, as noted by Walshe, present no symptoms pointing to intrathoracic disease. Among the more important symptoms are pain, particularly when the pleura is involved; dyspnœa, which is apt to be paroxysmal when due to pressure upon the trachea; cough, which may be dry and painful and accompanied by the expectoration of a dark mucoid sputum. This so-called prune-juice expectoration, which was present ten times in eighteen cases of primary cancer of the lung, was thought by Stokes to be of great diagnostic value.

In many instances there are signs of compression of the large veins, producing lividity of the face and upper extremities, or occasionally of only one arm. Compression of the trachea and bronchi may give rise to urgent dyspnœa. The heart may be pushed over to the opposite side. The pneumogastric and recurrent laryngeal nerves are occasionally involved in the growth.

Physical Signs.—The patient, according to Walshe, usually lies on the affected side. On inspection this side may be enlarged and immobile and the intercostal spaces are obliterated. This is more commonly due to the effusion than to the growth itself. The external lymph-glands may be enlarged, particularly the clavicular. The signs, on percussion and auscultation, are varied, depending much upon the presence or absence of fluid. Signs of consolidation are, of course, present; the tactile fremitus is absent and the breath-sounds are usually diminished in intensity. Occasionally there is typical bronchial breathing. Among other symptoms may be mentioned fever, which is present in a certain number of cases. Emaciation is not necessarily extreme. The duration of the disease is from six to eight months. Occasionally the disease runs a very acute course, as noted by Carswell. Cases are reported in which death occurred in a month or six weeks, and in one instance—Jaccoud—the patient died in a week from the onset of the symptoms.

Diagnosis.—In secondary growths this is not difficult. The development of pulmonary symptoms within a year or two after the removal of a cancer of the breast, or after the amputation of a limb for osteo-sarcoma, or the onset of similar symptoms in connection with cancer of the liver, or of the uterus, or of the rectum, would be extremely suggestive. In primary cases the unilateral involvement, the anomalous character of the physical signs, the occurrence of prune-juice expectoration, the progressive wasting, and the secondary involvement of the cervical glands are the important points in the diagnosis.

V. DISEASES OF THE PLEURA.

I. ACUTE PLEURISY.

Anatomically, the cases may be divided into dry or adhesive pleurisy and pleurisy with effusion. Another classification is into primary or secondary forms. According to the course of the disease, a division may be made into *acute* and *chronic* pleurisy, and as it is impossible, at present, to group the various forms etiologically, this is perhaps the most satisfactory division. The following forms of acute pleurisy may be considered:

I. Fibrinous or Plastic Pleurisy.

In this the pleural membrane is covered by a sheeting of lymph of variable thickness, which gives it a turbid, granular appearance, or the fibrin may exist in distinct layers. It occurs (1) as an independent affection, following cold or exposure. This form of acute plastic pleurisy without fluid exudate is not common in perfectly healthy individuals. Cases are met with, however, in which the disease sets in with the usual symptoms of pain in the side and slight fever, and there are the physical signs of pleurisy as indicated by the friction. After persisting for a few days, the friction murmur disappears and no exudation occurs. Union takes place between the membranes, and possibly the pleuritic adhesions which are found in such a large percentage of all bodies examined after death originate in these slight fibrinous pleurisies.

Fibrinous pleurisy occurs (2) as a secondary process in acute diseases of the lung, such as pneumonia, which is always accompanied by a certain amount of pleurisy, usually of this form. Cancer, abscess, and gangrene also cause plastic pleurisy when the surface of the lung becomes involved. This condition is specially associated in a large number of cases with tuberculosis. Pleural pain, stitch in the side, and a dry cough, with marked friction sounds on auscultation are the initial phenomena in many instances of phthisis. The signs are usually basic, but Burney Yeo has recently called attention to the frequency with which they occur at the apex.

II. Sero-fibrinous Pleurisy.

In a majority of cases of inflammation of the pleura there is, with the fibrin, a variable amount of fluid exudate, which produces the condition known as pleurisy with effusion.

Etiology.—For generations physicians have considered cold the potent factor in inducing pleurisy. This may be true in many cases, but modern views of serous inflammations scarcely recognize cold as anything more than a predisposing agent, which permits the action of various micro-organisms. We have not yet, however, brought all the acute pleu-

risies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or a chill. Of late years an attempt has been made, particularly by French writers, to show that the majority of acute pleurisies are tuberculous. In this connection the following facts may be admitted: (1) In a limited number of cases of pleurisy coming on abruptly in healthy persons the disease has been shown—(*a*) by post-mortem, in cases of accidental or sudden death, (*b*) by the subsequent history—to be tuberculous; (2) in a larger proportion of those cases which come on insidiously in persons who have been in failing health or who are delicate the disease is tuberculous from the outset; (3) the acute pleurisy, which occurs as a secondary, often a terminal, event in chronic affections, such as cirrhosis of the liver, Bright's disease, and cancer, is very frequently tuberculous. I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin. The subsequent history of cases of acute pleurisy forces us to conclude that in at least two thirds of the cases it is a curable affection. This may well be so, according to our present ideas of local tuberculous disease. One of the most interesting contributions to this question has been made from the records of Henry I. Bowditch, of Boston, to whom we are indebted for so many important contributions to our knowledge of pleurisy.* Of 90 cases of acute pleurisy which had been under observation between 1849 and 1879, 32 died of or had phthisis—a percentage large enough to indicate what an important *rôle* tuberculosis plays in the etiology of this disease.

Morbid Anatomy.—In sero-fibrinous pleurisy the serous exudate is abundant and the fibrin is found on the pleural surfaces and scattered through the fluid in the form of flocculi. The proportion of these constituents varies a great deal. In some instances there is very little membranous fibrin; in others it forms thick, creamy layers and exists in the dependent part of the fluid as whitish, curd-like masses. The fluid of sero-fibrinous pleurisy is of a ~~citron~~ color, either clear or slightly turbid, depending on the number of formed elements. In some instances it has a dark-brown color. The microscopical examination of the fluid shows leucocytes, occasional swollen cells, which may possibly be derived from the pleural endothelium, shreds of fibrillated fibrin, and a variable number of red blood-corpuscles. On boiling, the fluid is found to be rich in albumen. Sometimes it coagulates spontaneously. Its composition closely resembles that of blood-serum. Cholesterin, uric acid, and sugar are occasionally found. The amount of the effusion varies from a half to four litres.

The lung in acute sero-fibrinous pleurisy is more or less compressed. If the exudation is limited the lower lobe alone is atelectatic; but in an extensive effusion which reaches to the clavicle the entire lung will be found

* Vincent Y. Bowditch, in Boston Medical and Surgical Journal, 1889.

sions an increase in volume, which may appear to be much more than it really is as determined by mensuration. The intercostal spaces are obliterated. In right-sided effusions the apex beat may be lifted to the fourth interspace or be pushed beyond the left nipple, or may even be seen in the axilla. When the exudation is on the left side the heart's impulse may not be visible; but if the effusion is large it is seen in the third and fourth spaces on the right side, and sometimes as far out as the nipple, or even beyond it.

Palpation enables us more successfully to determine the deficient movements on the affected side, and the obliteration of the intercostal spaces, and more accurately to define the position of the heart's impulse. In simple sero-fibrinous effusion there is rarely any œdema of the chest walls. It is scarcely ever possible to obtain fluctuation. Tactile fremitus is greatly diminished or abolished. If the effusion is slight there may be only enfeeblement. The absence of the voice vibrations in effusions of any size constitutes one of the most valuable of physical signs. In children there may be much effusion with retention of fremitus. In rare cases the vibrations may be communicated to the chest walls through localized pleural adhesions.

Mensuration.—With the cyrtometer, if the effusion is excessive, a difference of from half an inch to an inch, or even, in large effusions, an inch and a half, may be found between the two sides. Allowance must be made for the fact that the right side is naturally larger than the left. With the saddle-tape the difference in expansion between the two sides can be conveniently measured.

Percussion.—Early in the disease, when the pain in the side is severe and the friction murmur evident, there may be no alteration, but with the gradual accumulation of the fluid the resonance becomes defective, and finally gives place to absolute dulness. From day to day the gradual increase in height of the fluid may be studied. In a pleuritic effusion rising to the fourth rib in front, the percussion signs are usually very suggestive. In the subclavicular region the attention is often aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion. The dulness has a peculiarly resistant, wooden quality, differing from that of pneumonia and readily recognized by skilled fingers. It has long been known that when the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind than it is in front, forming a parabola. Ellis and Garland, of Boston, who have made a careful study of this question, state that the line of dulness from behind forward may sometimes be represented by a curved line resembling the letter S. The condition is fully considered in Garland's exhaustive work on Pneumo-dynamics.

On the right side the dulness passes without change into that of the liver. On the left side in the nipple line it extends to and may obliterate Traube's semilunar space. If the effusion is moderate, the phenomenon of movable dulness may be obtained by marking carefully, in the sitting posture, the upper limit in the mammary region, and then in the recumbent posture, noting the change in the height of dulness. This infallible sign of fluid cannot always be obtained. In very copious exudation the dulness may reach the clavicle and even extend beyond the sternal margin of the opposite side.

Auscultation.—Early in the disease a friction rub can usually be heard, which disappears as the fluid accumulates. It is a to-and-fro dry rub, close to the ear, and has a leathery, creaking character. There is another pleural friction sound which closely resembles, and is scarcely to be distinguished from, the fine crackling crepitus of pneumonia. This may be heard at the commencement of the disease, and also, as pointed out in 1844 by MacDonnell, Sr., of Montreal, when the effusion has receded and the pleural layers come together again.

With even a slight exudation there is weakened or distant breathing. Often inspiration and expiration are distinctly audible, though distant, and have a tubular quality. Sometimes only a puffing tubular expiration is heard, which may have a metallic or amphoric quality. Loud resonant râles accompanying this may forcibly suggest a cavity. These pseudo-cavernous signs are met with more frequently in children, and often lead to error in diagnosis. Above the line of dulness the breath-sounds are usually harsh and exaggerated, and may have a tubular quality.

The vocal resonance is usually diminished or absent. The whispered voice is said to be transmitted through a serous and not through a purulent exudate (Bacelli's sign). There may, however, be intensification—bronchophony. The voice sometimes has a curious nasal, squeaking character, which was termed by Laennec *ægophony*, from its supposed resemblance to the bleating of a goat. In typical form this is not common, but it is by no means rare to hear a curious twang-like quality in the voice, particularly at the outer angle of the scapula.

In the examination of the heart in cases of pleuritic effusion it is well to bear in mind that when the apex of the heart lies beneath the sternum there may be no impulse. The determination of the situation of the organ may rest with the position of maximum loudness of the sounds. In the displaced organ a systolic murmur may be heard. When the lappet of lung over the pericardium is involved on either side there may be a pleuro-pericardial friction.

The *course* of acute sero-fibrinous pleurisy is very variable. After persisting for a week or ten days the fever subsides, the cough and pain disappear, and a slight effusion may be quickly absorbed. In cases in which the effusion reaches as high as the fourth rib recovery is usually slower. Many instances come under observation for the first time, after two or

three weeks' indisposition, with the fluid at a level with the clavicle. The fever may last from ten to twenty days without exciting anxiety, though, as a rule, in ordinary pleurisy from cold, as we say, the temperature in cases of moderate severity is normal within eight or ten days. Left to itself the natural tendency is to resorption; but this may take place very slowly. Even after it has persisted for months a sero-fibrinous exudate may completely disappear. With the absorption of the fluid there is a redux-friction crepitus, either leathery and creaking or crackling and râle-like, and for months, or even longer, the defective resonance and feeble breathing are heard at the base.

A sero-fibrinous exudate may persist for months without change, particularly in tuberculous cases, and will sometimes reaccumulate after aspiration and resist all treatment. The change of the exudate into pus will be spoken of in connection with empyema. Death is a rare termination of sero-fibrinous effusion. When one pleura is full and the heart is greatly dislocated the condition, although in a majority of cases producing remarkably little disturbance, is not without risk. *Sudden death* may occur, and its possibility under these circumstances should always be considered. I have seen two instances—one in right and the other in left sided effusion—both due, apparently, to syncope following slight exertion, such as getting out of bed. In neither case, however, was the amount of fluid excessive. Weil, who has studied carefully this accident, concludes as follows: (1) That it may be due to thrombosis or embolism of the heart or pulmonary artery, œdema of the opposite lung, or degeneration of the heart muscle; (2) such alleged causes as mechanical impediment to the circulation, owing to dislocation of the heart or twisting of the great vessels, require further investigation. It occurs more frequently in right than in left pleurisies, and the effusion is usually serous. Death may occur without any premonitory symptoms, usually during some movement or effort.

III. Purulent Pleurisy (*Empyema*).

Etiology.—Pus in the pleura is met with under the following conditions: (*a*) As a sequence of acute sero-fibrinous pleurisy. It is not always easy to say why, in certain cases, the exudate becomes purulent. It rarely does so in the acute pleurisies of healthy individuals. In children many cases are probably purulent from the outset. Aspiration, which is said to favor the occurrence of empyema, in my experience does so very rarely. (*b*) Purulent pleurisy is common as a secondary inflammation in various infectious diseases, among which scarlet fever takes the first place. It has long been known that the pleurisy supervening in the convalescence of this disease is almost always purulent. It should be remembered that it is latent in its onset, and that there may be no pulmonary symptoms. The pleurisy following typhoid fever is also usually purulent. Other infectious diseases—measles and whooping-cough

—are more rarely followed by this complication. Of late years especial attention has been paid to the connection of pneumonia with empyema, and it has been shown that very many cases come on insidiously either in the course of or during convalescence from this disease; and, lastly, a limited number of tuberculous pleurisies early become purulent. (*c*) Empyema results from local causes—fracture of the rib, penetrating wounds, malignant disease of the lung or œsophagus, and, perhaps most frequently of all, the perforation of the pleura by tuberculous cavities.

The bacteriology of empyema is of some importance. A sterile exudate suggests tuberculosis. In many cases the pneumococci are present, and these, as a rule, run a very favorable course. The streptococci are found most commonly in the secondary cases in connection with septic processes. In a few instances psorosperms have been found in the exudate.

Morbid Anatomy.—On opening an empyema post mortem, we usually find that the effusion has separated into a clear, greenish-yellow serum above and the thick, cream-like pus below. The fluid may be scarcely more than turbid, with flocculi of fibrin through it. In other instances it is uniformly thick and creamy, without any fibrin. It usually has a heavy, sweetish odor, but in some instances—particularly those following wounds—it is fetid. In cases of gangrene of the lung or pleura the pus has a horribly stinking odor. Microscopically it has the characters of ordinary pus. The pleural membranes are greatly thickened, and present a grayish-white layer from 1 to 2 mm. in thickness. On the costal pleura there may be erosions, and in old cases fistulous communications are common. The lung may be compressed to a very small limit, and the visceral pleura also may show perforations.

Symptoms.—Purulent pleurisy may begin abruptly, with the symptoms already described. More frequently it comes on insidiously in the course of other diseases or follows an ordinary sero-fibrinous pleurisy. There may be no pain in the chest, very little cough, and no dyspnœa, unless the side is very full. Symptoms of septic infection are rarely wanting. If in a child, there is a gradually developing pallor and weakness; sweats occur, and there is irregular fever. A cough is by no means constant.

Physical Signs.—Practically they are those already considered in pleurisy with effusion. There are, however, one or two additional points to be mentioned. In empyema, particularly in children, the disproportion between the sides may be extreme. The intercostal spaces may not only be obliterated, but may bulge. Much more frequently there is œdema of the chest walls. The network of subcutaneous veins may be very distinct. It must not be forgotten that in children the breath-sounds may be *loud and tubular* over a purulent effusion of considerable size. Whispered pectoriloquy is usually not heard in empyema (Bacelli's sign). The dislocation of the heart and the displacement of the liver are more marked

in empyema than in sero-fibrinous effusion—probably, as Senator suggests, owing to the greater weight of the fluid.

A curious phenomenon associated generally with empyema, but which may occur in the sero-fibrinous exudate, is *pulsating pleurisy*, first described by MacDonnell, Sr., of Montreal. Of 42 cases 39 occurred on the left side. In all but one case the fluid was purulent. Pneumothorax may be present. There are two groups of cases, the intrapleural pulsating pleurisy and the pulsating *empyema necessitatis*, in which there is an external pulsating tumor. No satisfactory explanation has been offered how the heart impulse is thus forcibly communicated through the effusion.

Empyema is a chronic affection, which in a few instances terminates naturally in recovery, but a majority of cases, if left alone, end in death. The following are some modes of natural cure: (*a*) By absorption of the fluid. In small effusions this may take place gradually. The chest wall sinks. The pleural layers become greatly thickened and enclose between them the inspissated pus, in which lime salts are gradually deposited. Such a condition may be seen once or twice a year in the post-mortem room of any large hospital. (*b*) By perforation of the lung. Although in this event death may take place rapidly, by inundation of the bronchial tubes, yet in many cases it occurs gradually and recovery follows. Since 1873, when I saw a case of this kind in Traube's clinic, and heard his remarks on the subject, I have seen a number of instances of the kind and can corroborate his statement as to the favorable termination of many of them. Empyema may discharge either by opening into the bronchus and forming a fistula or, as Traube pointed out, by producing necrosis of the pulmonary pleura, sufficient to allow the soakage of the pus through the spongy lung tissue into the bronchi. In the first way pneumothorax usually, though not always, develops. In the second way the pus is discharged without formation of pneumothorax. Even with a bronchial fistula recovery is possible. (*c*) By perforation of the chest wall—*empyema necessitatis*. This is by no means an unfavorable method, as many cases recover. The perforation may occur anywhere in the chest wall, but is, as Cruveilhier remarked, more common in front. It may be anywhere from the third to the sixth interspace, usually, according to Marshall, in the fifth. It may perforate in more than one place, and there may be a fistulous communication which opens into the pleura at some distance from the external orifice. The tumor, when near the heart, may pulsate. The discharge may persist for years. In Copeland's Dictionary is mentioned an instance of a Bavarian physician who had a pleural fistula for thirteen years and enjoyed fairly good health.

An empyema may perforate the neighboring organs, the œsophagus, peritonæum, pericardium, or the stomach. Very remarkable cases are those which pass down the spine and along the psoas into the iliac fossa, and simulate a psoas or lumbar abscess.

IV. Tuberculous Pleurisy.

This has already been considered. Here it is sufficient to say that it occurs as: (*a*) An acute affection, accompanied by abundant sero-fibrinous fluid. In this category come certainly a proportion of the cases regarded as acute pleurisy from cold. (*b*) As a subacute affection, latent in its origin and insidious in its course, frequently preceding the development of or coming on concurrently with pulmonary tuberculosis. (*c*) As an acute pleurisy, the result of direct extension from the lung in cases of well-marked phthisis, and in which the fluid may be either sero-fibrinous or purulent. (*d*) Chronic adhesive tuberculous pleurisy, which may be unilateral or bilateral, unaccompanied by exudation and characterized by great thickening of the pleural membranes, in which are tubercles and caseous masses of varying sizes.

The symptoms and physical signs of tuberculous pleurisy with exudation do not require any description other than that already given in connection with the sero-fibrinous and purulent forms.

V. Other Varieties of Pleurisy.

Hæmorrhagic Pleurisy.—A bloody effusion is met with under the following conditions: (*a*) In the pleurisy of asthenic states, such as cancer, Bright's disease, and occasionally in the malignant fevers. It is interesting to note the frequency with which hæmorrhagic pleurisy is found in cirrhosis of the liver. It occurred in the very patient in whom Laennec first accurately described this disease. While this may be a simple hæmorrhagic pleurisy, in a majority of the cases which I have seen it has been tuberculous. (*b*) Tuberculous pleurisy, in which the bloody effusion may result from the rupture of newly formed vessels in the soft exudate accompanying the eruption of miliary tubercles, or it may come from more slowly formed tubercles in a pleurisy secondary to extensive pulmonary disease. (*c*) Cancerous pleurisy, whether primary or secondary, is frequently hæmorrhagic. (*d*) Occasionally hæmorrhagic exudation is met with in perfectly healthy individuals, in whom there is not the slightest suspicion of tuberculosis or cancer. In one such case, a large, able-bodied man, the patient was to my knowledge healthy and strong eight years afterward. And, lastly, it must be remembered that during aspiration the lung may be wounded and blood in this way get mixed with the sero-fibrinous exudate. The condition of hæmorrhagic pleurisy is to be distinguished from hæmothorax, due to the rupture of aneurism or the pressure of a tumor on the thoracic veins.

Diaphragmatic Pleurisy.—The inflammation may be limited partly or chiefly to the diaphragmatic surface. This is often a dry pleurisy, but there may be effusion, either sero-fibrinous or purulent, which is circumscribed on the diaphragmatic surface. In these cases the pain is low in

may simulate that of acute abdominal disease.

Intense subjective with trifling objective features are always suggestive

the zone of the diaphragm and, as Chéneau de Mussy pointed out, may be intensified by pressure at the point of insertion of the diaphragm at the tenth rib. The diaphragm is fixed and the respiration is thoracic and short. Andral noted in certain cases severe dyspnœa and attacks simulating angina. As mentioned, the effusion is usually plastic, not serous. Serous or purulent effusions of any size limited to the diaphragmatic surface are extremely rare.

Encysted Pleurisy.—The effusion may be circumscribed by adhesions or separated into two or more pockets or loculi, which communicate with each other. This is most common in empyema. In these cases there have usually been, at different parts of the pleura, multiple adhesions by which the fluid is limited. In other instances the recent false membranes may encapsulate the exudation on the diaphragmatic surface, for example, or the part of the pleura posterior to the mid-axillary line. The condition may be very puzzling during life, and present special difficulties in diagnosis. In some cases the tactile fremitus is retained along certain lines of adhesion. The exploratory needle should be freely used when there is any doubt.

Interlobar Pleurisy forms an interesting and not uncommon variety. In nearly every instance of acute pleurisy the interlobular serous surfaces are also involved and closely agglutinated together, and sometimes the fluid is encysted between them. In a recent case of this kind following pneumonia, there was between the lower and upper and middle lobes of the right side an enormous purulent collection, which looked at first like a large abscess of the lung. These collections may perforate the bronchi, and the cases present special difficulties in diagnosis.

Diagnosis of Pleurisy.—Acute plastic pleurisy is readily recognized. In the diagnosis of pleuritic effusion the first question is, Does a fluid exudate exist? the second, What is its nature? In large effusions the increase in the size of the affected side, the immobility, the absence of tactile fremitus, together with the displacement of organs, give infallible indications of the presence of fluid. The chief difficulty arises in effusions of moderate extent, when the dulness, the presence of bronchophony, and, perhaps, tubular breathing may simulate *pneumonia*. The chief points to be borne in mind are: (*a*) Differences in the onset and in the general characters of the two affections, more particularly the initial chill, the higher fever, more urgent dyspnœa, and the rusty expectoration, which characterize pneumonia. (*b*) Certain physical signs—the more wooden character of the dulness, the greater resistance, and the marked diminution or the absence of tactile fremitus in pleurisy. The auscultatory signs may be deceptive. It is usually, indeed, the persistence of tubular breathing, particularly the high-pitched, even amphoric expiration, heard in some cases of pleurisy, which has raised the doubt. The intercostal spaces are more commonly obliterated in pleuritic effusion than in pneumonia. As already mentioned, the displacement of organs is a very valuable sign.

Nowadays with the hypodermic needle the question is easily settled. A separate small syringe with a capacity of two drachms should be reserved for exploratory purposes, and the needle should be longer and firmer than in the ordinary hypodermic instrument. With careful preliminary disinfection the instrument can be used with impunity, and in cases of doubt the exploratory puncture should be made without hesitation. I have never seen the slightest ill effects follow its use. Cases are reported of pneumothorax resulting from it, but they are extremely rare. The hypodermic needle is especially useful in those cases in which there are pseudo-cavernous signs at the base. In cases, too, of massive pneumonia, in which the bronchi are plugged with fibrin, if the patient has not been seen from the outset, the diagnosis may be impossible without it.

On the left side it may be difficult to differentiate a very large pericardial from a pleural effusion. The retention of resonance at the base, the presence of Skoda's resonance toward the axilla, the absence of dislocation of the heart-beat to the right of the sternum, the feebleness of the pulse and of the heart-sounds, and the urgency of the dyspnœa, out of all proportion to the extent of the effusion, are the chief points to be considered. Unilateral hydrothorax, which is not at all uncommon in heart-disease, presents signs identical with those of sero-fibrinous effusion. Certain tumors within the chest may simulate pleural effusion. It should be remembered that many intrathoracic growths are accompanied by exudation. Malignant disease of the lung and of the pleura and hydatids of the pleura produce extensive dulness, with suppression of the breath-sounds, simulating closely effusion.

On the right side abscess of the liver and hydatid cysts may rise high into the pleura and produce dulness and enfeebled breathing. Often in these cases there is a friction sound, which should excite suspicion, and the upper outline of the dulness is sometimes plainly convex. In all these instances the exploratory puncture should be made.

The second question, as to the nature of the fluid, is quickly decided by the use of the needle. The persistent fever, the occurrence of sweats, and the increase in the pallor suggest the presence of pus. In children the complexion is often sallow and earthy. The unexpected, however, often happens, and repeatedly, in protracted cases, even in children, when the general symptoms and the appearance of the patient has been most strongly suggestive of pus, the syringe has withdrawn clear fluid. On the other hand, effusions of short duration may be purulent, even when the general symptoms do not suggest it. The following statement may be made with reference to the prognostic import of the bacteriological examination of the aspirated fluid: The presence of the pneumococcus is of favorable significance, as such cases usually get well rapidly, even with a single aspiration. The pus organisms—staphylococci and streptococci—are more common in empyema of septic origin, and such cases are notori-

ously less hopeful than others. A sterile fluid indicates in a majority of instances a tuberculous origin.

Treatment.—At the onset the severe pain may demand leeches, which usually give relief, but a hypodermic of morphia is more effective. The Paquelin cautery may be lightly but freely applied. It is well to administer a mercurial or saline purge. Fixing the side by careful strapping with long strips of adhesive plaster, which should pass well over the middle line, drawn tightly and evenly, gives great relief, and I can corroborate the statement of F T. Roberts as to its efficacy. Cupping, wet or dry, is now seldom employed. Blisters are of no special service in the acute stages, although they relieve the pain. The ice-bag may be used as in pneumonia. The general treatment of the early stage should be rest in bed and a liquid diet. Medicines are rarely required. A Dover's powder may be given at night. Mercurials are not indicated.

When the effusion has taken place, mustard plasters or iodine, producing slight counter-irritation, appear useful, particularly in the later stages. The following rational plan is successful in some cases. It is based upon the idea that if the blood serum is depleted or if it is kept concentrated, the liquid will be absorbed from the lymph spaces, of which the pleura is one, to equalize the loss. To do this the patient should have the daily amount of liquid food greatly restricted. If there is no fever, a meat diet, with an egg and dry bread and eight to ten ounces of liquid in the form of milk or water, should be given. Salt articles of food may be used, but I do not think it necessary to give, as some do, doses of salt. The second element in the treatment is the active depletion of blood serum, which is effected in the way introduced by Matthew Hay. Every morning, if the patient is robust, otherwise every second morning, from half an ounce to an ounce and a half of Epsom salts is given an hour before breakfast, in as concentrated a form as is possible. This produces copious liquid discharges. I have seen large exudations disappear rapidly when this plan was followed. By acting upon the skin and kidneys, the same end may be obtained, but with much less certainty. The vapor or hot bath may be used and an occasional dose of pilocarpin. Diuretics, such as digitalis, squills, and acetate of ~~lead~~, may sometimes be required. I rarely resort, however, to diuretics or diaphoretics in the treatment of pleurisy with effusion. Iodide of potassium is of doubtful benefit.

Aspiration of the fluid is the most thorough and satisfactory method and should be resorted to whenever the effusion becomes large or if it resists the ordinary methods of treatment. The credit of introducing aspiration in pleuritic effusions is due to Morrill Wyman, of Cambridge, Mass., and Henry I. Bowditch, of Boston. Years prior to Dieulafoy's work, aspiration was in constant use at the Massachusetts General Hospital and was advocated repeatedly by Bowditch. As the question is one of some historical interest, I give the author's conclusions concerning aspiration, expressed more than forty years ago, and which practically represent

the opinion of clinical physicians to-day: "(1) The operation is perfectly simple, but slightly painful, and can be done with ease upon any patient in however advanced a stage of the disease. (2) It should be performed forthwith in *all* cases in which there is complete filling up of one side of the chest. (3) He had determined to use it in *any* case of even *moderate* effusion lasting more than a few weeks and in which there should seem to be an indisposition to resist ordinary modes of treatment. (4) He urged this practice upon the profession as a very important measure in practical medicine; believing that by this method death may frequently be prevented from ensuing either by sudden attack of dyspnœa or subsequent phthisis, and, finally, from the gradual wearing out of the powers of life or inability to absorb the fluid. (5) He believed that this operation would sometimes prevent the occurrence of those tedious cases of spontaneous evacuation of purulent fluid and those great contractions of the chest which occur after long-continued effusion and the subsequent discharge or absorption of a fluid."

There is scarcely anything to be added to-day to these observations. When the fluid reaches to the clavicle the indication for aspiration is imperative, even though the patient be comfortable and present no signs of pulmonary distress. The presence of fever is not a contra-indication; indeed, sometimes with serous exudates the temperature falls after aspiration.

The operation is extremely simple and is practically without risk. The spot selected for puncture should be either in the seventh interspace in the mid-axilla or at the outer angle of the scapula in the eighth interspace. The arm of the patient should be brought forward with the hand on the opposite shoulder, so as to widen the interspaces. The needle should be thrust in close to the upper margin of the rib, so as to avoid the intercostal artery, the wounding of which, however, is an excessively rare accident. The fluid should be withdrawn slowly. The amount will depend on the size of the exudate. If the fluid reaches to the clavicle a litre or more may be withdrawn with safety.

During aspiration if the patient feels faint it is best to interrupt the operation, for sudden death has occasionally happened during the withdrawal. It is, however, a much less common accident than sudden death in cases of full pleura without operation. Cough is a symptom which frequently develops toward the close of aspiration. Though very painful it need not excite alarm. French writers have described cases of albuminous expectoration, associated with dyspnœa, which may come on after the tapping and prove rapidly fatal. It must be an excessively rare complication. The conversion of a sero-fibrinous into a purulent fluid is a danger which need not be considered. I have never met with an instance of the kind.

Empyema is really a surgical affection, and I shall make only a few general remarks upon its treatment. When it has been determined by

Albuminous expectoration — see under Hayem and Sessier in Revue de Medecine, 1889, p. 33.

Case of Laimé about 1 3/4 pints in 24 hrs

exploratory puncture that the fluid is purulent, aspiration should not be performed, except as preliminary to operation or as a temporary measure. Perhaps it is better not to have an exception to this rule, although the empyemas of children and the pneumonic empyema occasionally get well rapidly after a single tapping. It is sad to think of the number of lives which are sacrificed annually by the failure to recognize that empyema should be treated as an ordinary abscess, by free incision. The operation dates from the time of Hippocrates and is by no means serious. A majority of the cases get well, providing that free drainage is obtained, and it makes no difference practically what measures are followed so long as this indication is met. The good results in any method depend upon the thoroughness with which the cavity is drained. Irrigation of the cavity is rarely necessary unless the contents are fetid. Sudden collapse has happened during irrigation and a remarkable accident is the occurrence of convulsions. In the subsequent treatment a point of great importance in facilitating the closure of the cavity is the distention of the lung on the affected side. This may be accomplished by the method advised by Walter James, which has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily, for a certain length of time, increasing gradually with the increase of his strength, transfers by air-pressure water from one bottle to another. The bottles should be large, holding at least a gallon each, and by the arrangement of tubes, as in the Wolff's bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess cavity is gradually closed, partly by the falling in of the chest wall and partly by the expansion of the lung. In some instances it is necessary to resect portions of one or more ribs.

The physician is often asked, in cases of empyema with emaciation, hectic and feeble rapid pulse, whether the patient could stand the operation. Even in the most desperate cases the surgeon should never hesitate to make a free incision.

II. CHRONIC PLEURISY.

This affection occurs in two forms: (1) *Chronic pleurisy with effusion*, in which the disease may set in insidiously or may follow an acute serofibrinous pleurisy. There are cases in which the liquid persists for months without undergoing any special alteration and without becoming purulent. Such cases have the characters which we have described under pleurisy with effusion. (2) *Chronic dry pleurisy.* The cases are met with (*a*) as a sequence of ordinary pleural effusion. When the exudate is absorbed and the layers of the pleura come together there is left between them a variable amount of fibrinous material which gradually undergoes organi-

zation, and is converted into a layer of firm connective tissue. This process goes on at the base, and is represented clinically by a slight grade of flattening, deficient expansion, defective resonance on percussion, and enfeebled breathing. After recovery from empyema the flattening and retraction may be still more marked. In both cases it is a condition which can be greatly benefited by pulmonary gymnastics. In these firm, fibrous membranes calcification may occur, particularly after empyema. It is not very uncommon to find between the false membranes a small pocket of fluid forming a sort of pleural cyst. In the great majority of these cases the condition is one which need not cause anxiety. There may be an occasional dragging pain at the base of the lung or a stitch in the side, but patients may remain in perfectly good health for years. The most advanced grade of this secondary dry pleurisy is seen in those cases of empyema which have been left to themselves and have perforated and ultimately healed by a gradual absorption or discharge of the pus, with retraction of the side of the chest and permanent carnification of the lung. Traumatic lesions, such as gunshot wounds, may be followed by an identical condition. Post mortem, it is quite impossible to separate the layers of the pleura, which are greatly thickened, particularly at the base, and surround a compressed, airless, fibroid lung.

(*b*) *Primitive dry pleurisy.* This condition may directly follow the acute plastic pleurisy already described; but it may set in without any acute symptoms whatever, and the patient's attention may be called to it by feeling the pleural friction. A constant effect of this primitive dry pleurisy is the adhesion of the layers. This is probably an invariable result, whether the pleurisy is primary or secondary. The organization of the thin layer of exudation in a pneumonia will unite the two surfaces by delicate bands. Pleural adhesions are extremely common, and it is rare to examine a body entirely free from them. They may be limited in extent or universal. Thin fibrous adhesions do not produce any alteration in the percussion characters, and, if limited, there is no special change heard on auscultation. When, however, there is general synechia on both sides the expansile movement of the lung is considerably impaired. We should naturally think that universal adhesions would interfere materially with the function of the lungs, but practically we see many instances in which there has not been the slightest disturbance. The physical signs of total adhesion are by no means constant. It has been stated that there is a marked disproportion between the degree of expansion of the chest walls and the intensity of the vesicular murmur, but the latter is a very variable factor, and under perfectly normal conditions the breath-sounds, with very full chest expansion, may be extremely feeble.

Is there a primitive dry pleurisy which gradually leads to great thickening of the membranes, and which ultimately may invade the lung and induce cirrhotic change? Upon this question neither pathologists nor clinicians agree. I think that Sir Andrew Clark, in his Lumleian lectures

at the Royal College of Physicians (1885), has made good his claim that such a disease does exist. At the outset in these cases there is a dry pleurisy, usually at one base, indicated by the usual signs; and this persists in spite of all treatment. There is no evidence of fluid; the general health may not be much impaired, or there may be slight fever and disturbed digestion. The cases give great anxiety, owing to the natural suspicion that tuberculosis exists. In time the evidence of dulness is found at the base. There are feeble breathing and creaking, leathery friction sounds. There may be commencing retraction of the side. Clinically these cases are of great interest, and should, I think, be separated, on the one hand, from the condition which follows a healed empyema or old pleurisy with effusion, and, on the other, from the rare instances of primitive cirrhosis of the lung. However, in all three states there may ultimately be an almost identical clinical picture. Anatomically in these pleuritic cases the pleura, particularly that surrounding the lower lobe, sometimes the entire membrane, is thickened, the two layers are intimately united, and fibrinous bands passing from the pleura traverse the lung tissue, sometimes dividing it in a remarkable way into sections. The bronchi may present marked dilatations, though this is not always the case, and the lung tissue is more or less sclerosed. The cases belong to the group of chronic pneumonias called by Charcot pleurogenous. In many instances there can be no question as to their non-tuberculous nature. There are cases, however, in which, with chronic pleurogenous pneumonia in the lower lobe, there are cavity formations at the apex and tuberculous lesions in other parts. Such may, of course, be tuberculous from the outset.

Lastly, there is a primitive dry pleurisy of tuberculous origin. In it both parietal and costal layers are greatly thickened—perhaps from two to three millimetres each—and present firm fibroid, caseous masses and small tubercles, while uniting these two greatly thickened layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. This may be a local process confined to one pleura, or it may be in both. I have seen two typical instances of it—one in a young, well-nourished Irish girl, who died of malignant scarlet fever, in whom one pleura was in the condition above described, and there were no other tuberculous lesions. The other was in a young man who died of typhoid fever, in whom both pleuræ were uniformly thickened and tuberculous without any fluid exudate. These cases are sometimes associated with a similar condition of the pericardium and peritonæum.

Occasionally remarkable vaso-motor phenomena occur in chronic pleurisy, whether simple or in connection with tuberculosis of an apex. Flushing or sweating of one cheek or dilatation of the pupil are the common manifestations. They appear to be due to involvement of the first thoracic ganglion at the top of the pleural cavity.

III. HYDROTHORAX.

Hydrothorax is a transudation of simple non-inflammatory fluid into the pleural cavities, and occurs as a secondary process in many affections. The fluid is clear, without any flocculi of fibrin, and the membranes are smooth. It is met with more particularly in connection with general dropsy, either renal, cardiac, or hæmic. It may, however, occur alone, or with only slight œdema of the feet. A child was admitted to the Montreal General Hospital with urgent dyspnœa and cyanosis, and died the night after admission. She had extensive bilateral hydrothorax, which had come on early in the nephritis of scarlet fever. In renal disease hydrothorax is almost always bilateral, but in heart affections one pleura is more commonly involved. The physical signs are those of pleural effusion, but the exudation is rarely excessive. In kidney and heart disease, even when there is no general dropsy, the occurrence of dyspnœa should at once direct attention to the pleura, since many patients are carried off by a rapid effusion. Post-mortem records show the frequency with which this condition is overlooked. The saline purges will in many cases rapidly reduce the effusion, but, if necessary, aspiration should repeatedly be practised.

IV. PNEUMOTHORAX (*Hydro-Pneumothorax and Pyo-Pneumothorax*).

Air alone in the pleural cavity, to which the term pneumothorax is strictly applicable, is an extremely rare condition. It is almost invariably associated with a serous fluid—hydro-pneumothorax, or with pus—pyo-pneumothorax.

Etiology.—It has usually been taught that there is an inherent tendency to pneumothorax, which is induced as soon as the pleura is opened. The experiments of S. West seem, however, to indicate the existence of a coherent force between the pleural surfaces much in excess of the elasticity of the lung, and sufficient in certain instances to maintain these organs in contact with the thoracic wall, even when there is free access to the pleura; so that in reality force is required to overcome the normal adhesion between the pleural membranes.

Pneumothorax arises: (1) In perforative wounds of the chest, in which case it is sometimes associated with extensive cutaneous emphysema. It has followed exploratory puncture with a hypodermic needle, as in two cases reported by Herman Biggs. Pneumothorax rarely follows fracture of the rib, even though the lung may be torn. (2) In perforation of the pleura through the diaphragm, usually by malignant disease of the stomach or colon. The pleura may also be perforated in cases of cancer of the œsophagus. (3) When the lung is perforated. This is by far the most common cause, and may occur: (*a*) In a normal lung from rupture

of the air-vesicles during straining. Special attention has lately been called to this accident by S. West and De H. Hall. The air may be absorbed and no ill effect follows. It does not necessarily excite pleurisy, as pointed out many years ago by Gairdner, but inflammation and effusion are the usual result. (*b*) From perforation due to local disease of the lung, either the softening of a caseous focus or the breaking of a tuberculous cavity. According to S. West, ninety per cent of all the cases are due to this cause. Less common are the cases due to septic bronchopneumonia and to gangrene. A rare cause is the breaking of a hæmorrhagic infarct in chronic heart-disease, of which I met an instance a few years ago. (*c*) Perforation of the lung from the pleura, which arises in certain cases of empyema and produces a pleuro-bronchial fistula.

Pneumothorax occurs chiefly in adults, though cases are met with in very young children. It is more frequent in males than in females.

Morbid Anatomy.—If a trocar or blow-pipe is inserted between the ribs, there may be a jet of air of sufficient strength to blow out a lighted match. On opening the thorax the mediastinum and pericardium are seen to be pushed, or rather, as Douglas Powell pointed out, drawn over to the opposite side; but, as before mentioned, the heart is not rotated, and the relation of its parts is maintained much as in the normal condition. A serous or purulent fluid is usually present, and the membranes are inflamed. The cause of the pneumothorax can usually be found without difficulty. In the great majority of instances it is the perforation of a tuberculous cavity or a breaking of a superficial caseous focus. The orifice of rupture may be extremely small. In chronic cases there may be a fistula of considerable size communicating with the bronchi. The lung is usually compressed and carnified.

Symptoms.—The onset is usually sudden and characterized by severe pain in the side, urgent dyspnœa, and signs of general distress, as indicated by slight lividity and a very rapid and feeble pulse. There may, however, be no urgent symptoms, particularly in cases of long-standing phthisis. On more than one occasion I have found, post mortem, a pneumothorax which was unsuspected during life. West states that even in healthy adults this latent pneumothorax may occasionally occur.

The *physical signs* are very distinctive. *Inspection* shows marked enlargement of the affected side with immobility. The heart impulse is usually much displaced. On *palpation* the fremitus is greatly diminished or more commonly abolished. On *percussion* the resonance may be tympanitic or even have an amphoric quality. This, however, is not always the case. It may be a flat tympany, resembling Skoda's resonance. In some instances it may be a full, hyperresonant note, like emphysema; while in others—and this is very deceptive—there is dulness. These extreme variations depend doubtless upon the degree of intrapleural tension. On several occasions I have known an error in diagnosis to result from ignorance of the fact that, in certain instances, the percussion note

may be "muffled, toneless, almost dull" (Walshe). There is usually dulness at the base from effused fluid, which can readily be made to change the level by altering the position of the patient. Movable dulness can be obtained much more readily in pneumothorax than in a simple pleurisy. On *auscultation* the breath-sounds are suppressed. Sometimes there is only a distant feeble inspiratory murmur of marked amphoric quality. The contrast between the loud exaggerated breath-sounds on the normal side and the absence of the breath-sounds on the other is very snggestive. The râles have a peculiar metallic quality, and on coughing or deep inspiration there may be what Laennec termed the metallic tinkling. The voice, too, has a curious metallic echo. What is sometimes called the coin-sound, termed by Trousseau the *bruit d'airain*, is very characteristic. To obtain it the auscultator should place one ear on the back of the chest wall while the assistant taps one coin on another on the front of the chest. The metallic echoing sound which is produced in this way is one of the most constant and characteristic signs of pneumothorax. And, lastly, the Hippocratic succussion may be obtained when the auscultator's head is placed upon the patient's chest and his body shaken. A splashing sound is produced, which may be audible at a distance. A patient may himself notice it in making abrupt changes in posture. Of other symptoms displacement of organs is most constant. As already mentioned, the heart may be drawn over to the opposite side, and the liver greatly displaced, so that its upper surface is below the level of the costal margin, a degree of dislocation never seen in simple effusion.

The *diagnosis* of pneumothorax rarely offers any difficulty, as the signs are very characteristic. In cases in which the percussion note is dull the condition may be mistaken for effusion. I made this mistake in a case of pulsating pleurisy, in which the pneumothorax followed heavy lifting, and it was not until several days later, after some of the fluid had been withdrawn, that a tympanitic note developed. Diaphragmatic hernia following a crush or other accident may closely simulate pneumothorax.

In cases of very large phthisical cavities with tympanitic percussion resonance and râles of an amphoric, metallic quality the question of pneumothorax is sometimes raised. In those rare instances of total excavation of one lung the amphoric and metallic phenomena may be most intense, but the absence of dislocation of the organs and of the succussion splash and of the coin sound suffice to differentiate this condition. Why the coin sound is not heard it is difficult to determine, unless its production is connected in some way with a certain degree of air-tension, which is not present in a vomica, however large. The condition of pyopneumothorax subphrenicus may simulate closely true pneumothorax.

The *prognosis* in cases of pneumothorax depends largely upon the cause. The phthisical cases usually die within a few weeks. Pneumothorax developing in a healthy individual often ends in recovery. There are cases of phthisis in which the pneumothorax, if occurring early, seems

to arrest the progress of the tuberculosis. This appeared to be the case in a man with chronic pneumothorax who was under my care in Philadelphia for between three and four years. It may be a chronic condition, as in the case just mentioned, and a fair measure of health may be enjoyed.

Treatment.—Practically these cases should be dealt with as ordinary pleurisy with effusion. Of course, when pneumothorax develops in advanced phthisis the indication is to relieve the pain and distress either by morphia or chloroform; but in cases which develop early the fluid should be withdrawn by aspiration, or, if purulent, permanent drainage should be obtained. Even when the condition has seemed to be most desperate I have known recovery to take place after thorough drainage of the sac. Portions of ribs may have to be excised, and during convalescence it is well for the patient to practise expansion of the lung in the manner already mentioned. There are cases of pneumothorax in phthisis in which the general condition is so good and the inconvenience so slight that to let well enough alone seems the best course. In such an occasional aspiration may be performed if the fluid increases. In some of the instances the mere tapping of the chest with a fine needle, so as to allow the escape of some of the air, seems to give relief by reducing the intrathoracic pressure. Good results are stated to have followed the method introduced by Potain, of replacing the air and fluid within the thorax by sterilized air.

AFFECTIONS OF THE MEDIASTINUM.

(1) **Simple Lymphadenitis.**—In all inflammatory affections of the bronchi and of the lungs the groups of lymph glands in the mediastinum become swollen. In the bronchitis of measles, for example, and in simple broncho-pneumonia the bronchial glands are large and infiltrated, the tissue is engorged and œdematous, sometimes intensely hyperæmic. Much stress has been laid by some writers on this enlargement of the glands in the posterior mediastinum, and De Mussy held that it was an important factor in inducing paroxysms of whooping-cough. They may attain a size sufficient to induce dulness beneath the manubrium and in the upper part of the interscapular regions behind, though this is often difficult to determine. In reality the glands lie chiefly upon the spine, and unless those which are deep in the root of the lung are large enough to induce compression of the adjacent lung tissue, I doubt if the ordinary bronchial adenopathy ever can be determined by percussion in the upper interscapular region. I have never met with an instance in which the compression of either bronchus seemed to have resulted from the glands, however large. Tuberculous affection of these glands has already been considered.

(2) **Suppurative Lymphadenitis.**—Occasionally abscess in the bronchial or tracheal lymph glands is found. It may follow the simple adenitis, but

is most frequently associated with the presence of tubercle. The liquid portion may gradually become absorbed and the inspissated contents undergo calcification. Serious accident occasionally occurs, as perforation into the œsophagus or into a bronchus.

(3) **Tumors; Cancer and Sarcoma.**—In Hare's elaborate study of 520 cases of disease of the mediastinum* there were 134 cases of cancer, 98 cases of sarcoma, 21 cases of lymphoma, 7 cases of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts, and instances of lipoma, gumma, and enchondroma. From this we see that cancer is the most common form of growth. The tumor occurred in the anterior mediastinum alone in 48 of the cases of cancer and 33 of the cases of sarcoma. The disease may be either primary in the mediastinal tissues and lymph structures or secondary. Sarcoma is more frequently primary than cancer. Males are more frequently affected than females. The age of onset is most commonly between thirty and forty.

Symptoms.—The signs of mediastinal tumor are those of intrathoracic pressure. *Dyspnœa* is one of the earliest and most constant symptoms, and may be due either to pressure on the trachea or on the recurrent laryngeal nerves. It may indeed be cardiac, due to pressure upon the heart or its vessels. In a few cases it results from the pleural effusion which so frequently accompanies intrathoracic growths. Associated with the dyspnœa is a cough, often severe and paroxysmal in character, with the brazen quality of the so-called aneurismal cough when a recurrent nerve is involved. The voice may also be affected from a similar cause. Pressure on the vessels is common. The superior vena cava may be compressed and obliterated, and when the process goes on slowly the collateral circulation may be completely effected. Less commonly the inferior vena cava or one or other of the subclavian veins is compressed. The arteries are much less rarely obstructed. It is remarkable how little the aorta may be involved, though entirely surrounded by a sarcomatous or cancerous mass. There may be dysphagia, due to compression of the œsophagus. In rare instances there are pupillary changes, either dilatation or contraction, due to involvement of the sympathetic.

Physical Signs.—On inspection there may be orthopnœa and marked cyanosis of the upper part of the body. In such instances, if of long duration, there are signs of collateral circulation and the superficial mammary and epigastric veins are enlarged. In a patient with Hodgkin's disease, at present under observation and in whom during the past sixteen months there has been progressive compression and now obliteration of the superior vena cava, the entire subcutaneous tissue of the front of the thorax seems a plexus of veins and the epigastric vessels are as large as the index-finger. Such instances are, I think, more common in lymphadenoma than in sarcoma or cancer. In these cases of chronic obstruction

* Fothergillian Prize Essay of the Medical Society of London, Philadelphia, 1889.

the finger-tips may be clubbed. There may be bulging of the sternum or the tumor may erode the bone and form a prominent subcutaneous growth. The rapidly growing lymphoid tumors more commonly than others perforate the chest wall. In four of thirteen cases of Hodgkin's disease, of which I have notes, there was mediastinal growth, and in three instances the sternum was eroded and perforated. The perforation may be on one side of the breast-bone. The projecting tumor may pulsate like an aneurism; the heart may be dislocated and its impulse much out of place. Contraction of one side of the thorax has been noted in a few instances. On palpation the fremitus is absent wherever the tumor reaches the chest wall. If pulsating, it rarely has the forcible, heaving impulse of an aneurismal sac. On auscultation there is usually silence over the dull region. The heart-sounds are not transmitted and the respiratory murmur is feeble or inaudible, rarely bronchial. Vocal resonance is, as a rule, absent. Signs of pleural effusion occur in a great many instances of mediastinal growth, and if in any doubt the aspirator needle should be used.

The *diagnosis* of mediastinal tumor from aneurism is sometimes extremely difficult. An interesting case reported and figured by Sokolosski, in Bd. 19 of the Deutsches Archiv für klinische Medicin, in which Oppolzer diagnosed aneurism and Skoda mediastinal tumor, illustrates how in some instances the most skilful of observers may be unable to agree. Scarcely a sign is found in aneurism which may not be duplicated in mediastinal tumor. This is not strange, since the symptoms in both are largely due to pressure. The time element is important. If a case has persisted for more than eighteen months the disease is probably aneurism. There are, however, exceptions to this. In the case of compression of the vena cava mentioned above, the disease has lasted for more than two years and the patient has improved so markedly under the use of arsenic that had he no other lymphatic enlargements the diagnosis might be uncertain. By far the most valuable sign of aneurism is the diastolic shock so often to be felt, and in a majority of cases to be heard, over the sac. This is rarely, if ever, present in mediastinal growths, even when they perforate the sternum and have communicated pulsation. Another point of importance is that in a tumor, advancing from the mediastinum, eroding the sternum and appearing externally, if aneurismal, has forcible, heaving, and distinctly expansile pulsations. The radiating pain in the back and arms and neck is rather in favor of aneurism, as is also a beneficial influence on it of iodide of potassium.

The frequency of pleural effusion in connection with mediastinal tumor is to be constantly borne in mind. It may give curiously complex characters to the physical signs—characters which are profoundly modified after aspiration of the liquid.

(4) **Abscess of the Mediastinum.**—Hare collected 115 cases of mediastinal abscess, in 77 of which there were details sufficient to permit the analysis. Of these cases the great majority occurred in males. Forty-four

were instances of acute abscess. The anterior mediastinum is most commonly the seat of the suppuration. The cases are most frequently associated with trauma. Some have followed erysipelas or occurred in association with eruptive fevers. Many cases, particularly the chronic abscesses, are of tuberculous origin. Of *symptoms*, pain behind the sternum is the most common. It may be of a throbbing character, and in the acute cases is associated with fever, sometimes with chills and sweats. If the abscess is large there may be dyspnœa. The pus may burrow into the abdomen, perforate through an intercostal space, or it may erode the sternum. Instances are on record in which the abscess has discharged into the trachea or œsophagus. In many cases, particularly of chronic abscess, the pus becomes inspissated and produces no ill effect. The *physical signs* may be very indefinite. A pulsating and fluctuating tumor may appear at the border of the sternum or at the sternal notch. The absence of *bruit*, of the diastolic shock, and of the expansile pulsation usually enables a correct diagnosis to be made. When in doubt a fine hypodermic needle may be inserted.

(5) **Miscellaneous Affections.**—In Hare's monograph there were 7 instances of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts, and cases of lipoma and gummata.

The *thymus gland* may be enlarged and produce the physical signs of mediastinal tumor. In children there are instances of spasm of the glottis, which is believed by some to depend upon enlargement of the thymus. Jacobi,* in his monograph, says that some instances of sudden death and also so-called thymic asthma may occasionally be referred to this cause. Malignant tumors of the thymus may attain considerable size and produce signs of tumor. In rare cases mediastinal growths develop from the *thyroid gland*. These may be substernal in position and directly connected with the gland. Kretschy has reported a sarcoma of the thyroid four and three quarter inches in length, which forms a mediastinal tumor passing to the level of the ninth dorsal vertebra. I have reported a somewhat similar instance, which developed in the left lobe of the thyroid and formed an elongated mass which passed down beside the trachea to the bifurcation.

(6) **Emphysema of the Mediastinum.**—Air in the cellular tissues of the mediastinum is met with in cases of trauma and occasionally in fatal cases of diphtheria and in whooping-cough. Champneys has called attention to its frequency in tracheotomy, in which he says the conditions favoring the production are division of the deep fascia, obstruction to the air-passages, and inspiratory efforts. The deep fascia, he says, should not be raised from the trachea. It is often associated with pneumothorax. The condition seems by no means uncommon. Angel Money found it in 16 of 28 cases of tracheotomy, and in two of these pneumothorax also was present.

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* Transactions of the Association of American Physicians, vol. iii.

Machine: Cobb

Beneke states that this may be due to compression of the trachea by the enlarged thymus when the head is bent back and calls attention to the danger of this in fat and ~~puffy~~ sickly infants. Brit. J. (Epitome) 4.14.94
See Paris Thesis 1884

It may extend to the subcutaneous tissues

Female this is mixed.

Gus Baillie

SECTION V.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

I. PERICARDITIS.

PERICARDITIS is the result of infective processes, primary or secondary, or arises by extension of inflammation from contiguous organs.

Etiology.—*Primary*, so-called idiopathic, inflammation of this membrane is rare; but cases are met with, most commonly in children, in which there is no evidence of rheumatism or other conditions with which the disease is usually associated.

Pericarditis from injury usually comes under the care of the surgeon in connection with the primary wound. Interesting cases are those in which the traumatism is from within, due to the passage of some foreign body—such as a needle, a pin, or a bone—through the œsophagus into the pericardium.

As a *secondary* process pericarditis is met with in the following affections: (*a*) A majority of the cases occur in connection with rheumatism. The percentage given by different authors ranges from thirty to seventy. The articular trouble may be slight or, indeed, the disease may be associated with acute tonsillitis of rheumatic subjects. Cases are recorded in which the pericarditis has preceded the articular disease. (*b*) Septic processes rank next to rheumatism. In the acute necrosis of bone and puerperal fever it is not uncommon. (*c*) Tuberculosis, in which the disease may be primary or part of a general involvement of the serous sacs or associated with extensive pulmonary disease. (*d*) Eruptive fevers. In children, the disease is not infrequent after scarlatina. It is rarely met with in measles, small-pox, or typhoid fever. In other infective diseases, such as diphtheria and pneumonia, it is rare. (*e*) Dyscrasias. Certain altered conditions of the system seem to render the pericardium more susceptible to inflammation. Of these gout takes the first place. In chronic Bright's disease pericarditis is by no means rare. The *pericardite brightique* of the French forms one of the most important groups of the disease in persons over fifty years of age, most frequently accom-

panying the chronic interstitial form. Pericarditis has been met with also in scurvy and diabetes.

Pericarditis by extension of disease from contiguous organs. In pleuro-pneumonia it forms one of the most serious complications, and was present in 5 cases in 100 post-mortems in this disease which I made at the Montreal General Hospital. It is most often met with in the pleuro-pneumonia of children and of alcoholics. The association with simple pleurisy is much less common. In ulcerative endocarditis, purulent myocarditis, and in aneurism of the aorta pericarditis is occasionally found. It may also result from extension of disease from the bronchial glands, the ribs, sternum, vertebræ, and even from the abdominal viscera.

Pericarditis occurs at all ages. Cases are reported in the fœtus. In the new-born it may result from septic infection through the navel. Throughout childhood the incidence of rheumatism and scarlet fever makes it a frequent affection, whereas late in life it is most often associated with Bright's disease and gout. Males are somewhat more frequently attacked than females. Climatic and seasonal influences have been mentioned by some writers. The so-called epidemics of pericarditis have been outbreaks of pneumonia with this as a frequent complication.

Anatomically as well as clinically the disease may be considered under the following divisions:

1. Acute, plastic, or dry pericarditis.
2. Pericarditis with effusion—sero-fibrinous, hæmorrhagic, or purulent.
3. Chronic adhesive pericarditis (adherent pericardium).

Acute Plastic Pericarditis.—This, the most common form, occurs usually as a secondary process, and is distinguished by the small amount of fluid exudation, which does not, as in the next variety, give special characters to the disease. It is a benign form and rarely, if ever, of itself proves fatal.

Anatomically it may be partial or general. In the mildest grades the serous membrane looks lustreless and roughened. This is due to the presence of a thin fibrinous sheeting, which can be lifted with the knife, showing the membrane beneath to be injected or in places ecchymotic. As the fibrinous sheeting increases in thickness the constant movement of the adjacent surfaces gives to it sometimes a ridge-like, at others a honeycombed appearance. With more abundant fibrinous exudation the membranes present an appearance resembling buttered surfaces which have been drawn apart. The fibrin is in long shreds, and the heart presents a curiously shaggy appearance—the so-called hairy heart of old writers—*cor villosum*.

In mild grades the subjacent muscle looks normal; but in the more prolonged and severe cases there is myocarditis, and for 2 or 3 mm. beneath the visceral layer the muscle presents a pale, turbid appearance.

Many of these acute cases are tuberculous; covered by the layers of lymph the granulations are easily overlooked in a superficial examination.

Slight fluid exudation is invariably present, entangled in the meshes of fibrin, but there may be very thick fibrinous layers without much serous effusion.

Symptoms.—The majority of cases of simple plastic pericarditis, like simple endocarditis, present no symptoms, and unless sought for there are no objective signs indicating its existence. In the post-mortem room it is not uncommon to find it in cases in which its presence has been unsuspected during life.

Pain is a variable symptom, not usually intense, and in this form rarely excited by pressure. It is more marked in the early stage, and may be referred either to the præcordia or to the region of the xiphoid cartilage. Instances are recorded of pain of an aggravated and most distressing character resembling angina. Fever is usually present, but it is not always easy to say how much depends upon the primary febrile affection, and how much upon the pericarditis. It is as a rule not high, rarely exceeding 102·5°. In rheumatic cases hyperpyrexia has been observed.

Physical Signs.—*Inspection* is negative; *palpation* may reveal the presence of a distinct fremitus caused by the rubbing of the roughened pericardial surfaces. This is usually best marked over the right ventricle. It is not always to be felt, even when the friction sound on auscultation is loud and clear. *Auscultation:* The friction sound, due to the movement of the pericardial surfaces upon each other, is one of the most distinctive of physical signs. It is double, corresponding to the systole and diastole; but the synchronism with the heart-sounds is not accurate, and the to-and-fro murmur usually outlasts the time occupied by the first and second sound. In rare instances the friction is single; more frequently it appears to be triple in character—a sort of canter rhythm. The sounds have a peculiar rubbing, grating quality, characteristic when once recognized, and rarely simulated by endocardial murmurs. Sometimes instead of grating there is a creaking quality—the *bruit de cuir neuf*—the new-leather murmur of the French. The pericardial friction appears superficial, very close to the ear, and is usually intensified by pressure with the stethoscope. It is best heard over the right ventricle, the part of the heart which is most closely in contact with the front of the chest—that is, in the fourth and fifth interspaces and adjacent portions of the sternum. There are instances in which the friction is most marked at the base over the aorta and at the superior reflection of the pericardium. Occasionally it is best heard at the apex. It may be limited and heard over a very narrow area, or it may be transmitted up and down the sternum. There are, however, no definite lines of transmission as in the endocardial murmur. An important point is the variability of sounds, both in position and quality; they may be heard at one visit and not at another. The maximum of intensity will be found to vary with position.

Diagnosis.—There is rarely any difficulty in determining the presence of a dry pericarditis, for the friction sounds are distinctive. The double murmur of aortic incompetency may simulate closely the to-and-fro pericardial rub. I recall one instance at least in which this mistake was made. The constant character of the aortic murmur, the direction of transmission, the phenomena in the arteries, and the associated conditions of the disease should be sufficient to prevent this error.

I have never known an instance in which pericarditis was mistaken for endocarditis, though writers refer to such, and give the differential diagnosis in the two affections. The only possible mistake could be made in those rare instances of single soft, systolic, pericardial friction.

Pleuro-pericardial friction is very common, and may be associated with endo-pericarditis, particularly in cases of pleuro-pneumonia. It is frequent, too, in phthisis. It is best heard over the left border of the heart, and is much affected by the respiratory movement. Holding the breath or taking a deep inspiration may annihilate it. The rhythm is not the simple to-and-fro diastolic and systolic, but the respiratory rhythm is superadded, usually intensifying the murmur during expiration and lessening it on inspiration. In phthisis there are instances in which, with the friction, a loud systolic click is heard, due to the compression of a thin layer of lung and the expulsion of a bubble of air from a small softening focus or from a bronchus.

Course and Termination.—Simple fibrinous pericarditis never kills, but it occurs so often in connection with serious affections that we have frequent opportunities to see all stages of its progress. In the majority of cases the inflammation subsides and the thin fibrinous laminæ gradually become converted into connective tissue, which unites the pericardial leaves firmly together. In other instances the inflammation progresses, with increase of the exudation, and the condition is changed from a "dry" to a "moist" pericarditis, or the pericarditis with effusion.

In a few instances—probably always tuberculous—the simple plastic pericarditis becomes chronic, and great thickening of both visceral and parietal layers is gradually induced.

Pericarditis with Effusion.—Though commonly a direct sequence of the dry or plastic pericarditis, of which it is sometimes ~~spoken as~~ the second stage, this form presents special features and deserves separate consideration. It is found most frequently in association with acute rheumatism, tuberculosis, and septicæmia, and sets in usually with the symptoms above described, namely, præcordial pain, with slight fever or a distinct chill.

In children the disease may, like pleurisy, come on without local symptoms, and, after a week or two of failing health, slight fever, shortness of breath, and increasing pallor, the physician may find, to his astonishment, signs of most extensive pericardial effusion. These latent cases are often tu-

Machine: Cobb

called.

berculous. The effusion may be sero-fibrinous, hæmorrhagic, or purulent. The amount varies from 200 or 300 c. c. to 2 litres. In the cases of sero-fibrinous exudation the pericardial membranes are covered with thick, creamy fibrin, which may be in ridges or honeycombed, or may present long, villous extensions. The parietal layer may be several millimetres in thickness and may form a firm, leathery membrane. The hæmorrhagic exudation is usually associated with tuberculous, or with cancerous pericarditis, or with the disease in the aged. The lymph is less abundant, but both surfaces are injected and often show numerous hæmorrhages. Thick, curdy masses of lymph are usually found in the dependent part of the sac. In the purulent effusion the fluid has a creamy consistency, particularly in tuberculosis. In many cases the effusion is really sero-purulent, a thin, turbid exudation containing flocculi of fibrin.

The pericardial layers are greatly thickened and covered with fibrin. When the fluid is pus, they present a grayish, rough, granular surface. Sometimes there are distinct erosions on the visceral membrane. The heart muscle in these cases becomes involved to a greater or less extent, and on section, the tissue, for a distance of from two to three millimetres, is pale and turbid, and shows evidence of fatty and granular change. Endocarditis coexists frequently, but rarely results from the extension of the inflammation through the wall of the heart.

Symptoms.—Even with copious effusion the onset and course may be so insidious that no suspicion of the true nature of the disease is aroused.

As in the simple pericarditis, pain may be present, either sharp and stabbing or as a sense of distress and discomfort in the cardiac region. It is more frequent with effusion than in the plastic form. Pressure at the lower end of the sternum usually aggravates it. Dyspnœa is a common and important symptom, one which, perhaps, more than any other, excites suspicion of grave disorder and leads to careful examination of heart and lungs. The patient is restless, lies upon the left side or, as the effusion increases, sits up in bed. Associated with the dyspnœa is in many cases a peculiarly dusky, anxious countenance. The pulse is rapid, small, sometimes regular, and may present the characters known as *pulsus paradoxus*, in which during each inspiration the pulse-beat becomes very weak or is lost. These symptoms are due, in great part, to the direct mechanical effect of the fluid within the pericardium which embarrasses the heart's action. Other pressure effects are distention of the veins of the neck, dysphagia, which may be a marked symptom, and irritative cough from compression of the trachea. Aphonia is not uncommon, due to compression or irritation of the recurrent laryngeal as it winds round the aorta. Another important pressure effect is exercised upon the left lung. In massive effusion the pericardial sac occupies such a large portion of the antero-lateral region of the left side that the condition has frequently been mistaken for pleurisy. Even in moderate grades the left

lung is somewhat compressed. This is an additional element in the production of the dyspnœa.

Great restlessness, insomnia, and in the later stages low delirium and coma are symptoms in the more severe cases. Delirium and marked cerebral symptoms are associated with the hyperpyrexia of rheumatic cases, but apart from the ordinary delirium there may be poculiar mental symptoms. The patient may become melancholic and show suicidal tendencies. In other cases the condition resembles closely delirium tremens. Sibson, who has specially described this condition, states that the majority of such cases recover. Chorea may also occur, as was pointed out by Bright. Epilepsy is a rare complication which has occurred, as in pleurisy, during paracentesis.

Physical Signs.—*Inspection.*—In children the præcordia bulges and with copious exudation the antero-lateral region of the left chest becomes enlarged. The intercostal spaces are prominent and there may be marked œdema of the wall. Perforation externally through a space is very rare. Owing to the compression of the lung, the expansion of the left side is greatly diminished. The diaphragm and left lobe of the liver may be pushed down and may produce a distinct prominence in the epigastric region.

Palpation.—A gradual diminution and final obliteration of the cardiac shock is a striking feature in progressive effusion. The apex beat is often raised an interspace and dislocated outward. Alteration in the position of the impulse simultaneously with the position of the patient, a sign upon which Oppolzer laid great stress, cannot often be determined, as the beat may, and usually does, disappear entirely. The pericardial friction may lessen with the effusion, though it often persists at the base when no longer palpable over the right ventricle, or may be felt in the erect and not in the recumbent posture. Fluctuation can rarely, if ever, be detected.

Percussion gives most important indications. The gradual distention of the pericardial sac pushes aside the margins of the lungs so that a large area comes in contact with the chest wall and gives a greatly increased percussion dulness. The form of this dulness is irregularly pear-shaped; the base or broad surface directed downward and the stem or apex directed upward toward the manubrium.

Auscultation.—The friction sound heard in the early stages may disappear when the effusion is copious, but often persists at the base or at the limited area of the apex. It may be audible in the erect and not in the recumbent posture. With the absorption of the fluid the friction returns. One of the most important signs is the gradual weakening of the heart-sounds, which with the increase in the effusion may become so muffled and indistinct as to be scarcely audible. The heart's action is usually increased and the rhythm disturbed. Occasionally a systolic endocardial murmur is heard.

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Important accessory signs in large effusion are due to pressure on the left lung. The antero-lateral margin of the lower lobe is pushed inside and in some instances compressed, so that percussion in the axillary region, in and just below the transverse nipple line, gives a modified percussion note, usually a flat tympany. Variations in the position of the patient may change materially this modified percussion area, over which on auscultation there is either feeble or tubular breathing.

Course.—Cases vary extremely in the rapidity with which the effusion takes place. In every instance, when a pericardial friction murmur has been detected, the practitioner should immediately outline with care—using the aniline pencil or nitrate of silver—the upper and left limits of cardiac dulness, since he will in this way have certain positive guides in determining the rate and grade of the effusion. In many instances the exudation is slight in amount, reaches a maximum within forty-eight hours, and then gradually subsides. In other instances the accumulation is more gradual and progressive, increasing for several weeks. To such cases the term chronic has been applied. The rapidity with which a sero-fibrinous effusion may be absorbed is surprising. The possibility of the absorption of purulent exudate is shown by the cases in which the pericardium contains semi-solid grayish masses in all stages of calcification. With sero-fibrinous effusion, if moderate in amount, recovery is the rule, with inevitable union, however, of the pericardial layers. In some of the septic cases there is a rapid formation of pus and a fatal result may follow in three or four days. More commonly, when death occurs with large effusion, it is not until the second or third week and takes place by gradual asthenia.

Prognosis.—In the sero-fibrinous effusions the outlook is good, and a large majority of all the rheumatic cases recover. The purulent effusions are, of course, more dangerous; the septic cases are usually fatal, and recovery is rare in the slow, insidious tuberculous forms.

Diagnosis.—Probably no serious disease is so frequently overlooked by the practitioner. Post-mortem experience shows how often pericarditis is present in cases which have not attracted attention. In a case of rheumatism, watched from the outset, with the attention directed daily to the heart, it is one of the simplest of diseases to diagnose; but when one is called to a case for the first time and finds perhaps an increased area of precordial dulness, it is often very hard to determine with certainty whether or not effusion is present.

The [illegible] heart and pericardial effusion. Although the differential signs are simple enough on paper, it is notoriously difficult in certain cases, particularly in short persons, to say which of the conditions exists. The points which deserve attention are:

(*a*) The character of impulse, which in dilatation, particularly in thin-chested people, is commonly visible and wavy.

see Roberts, Nervous Diseases, Epilepsy

Accentuation of the pulmonary second sound - Dock &c.

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The difficulty usually lies in distinguishing between dilatation of the heart and pericardial effusion. Although the differential signs are simple enough on paper, it is notoriously difficult in certain cases, particularly in stout persons, to say which of the conditions exists. The points which deserve attention are:

(*a*) The character of impulse, which in dilatation, particularly in thin-chested people, is commonly visible and wavy.

(*b*) The shock of the cardiac sounds is more distinctly palpable in dilatation.

(*c*) The area of dulness in dilatation rarely has the triangular form; nor does it, except in cases of mitral stenosis, reach so high along the left sternal margin or so ~~low~~ in the fifth and sixth interspaces *without visible or palpable impulse.* An upper limit of dulness shifting with the position speaks strongly for effusion.

(*d*) In dilatation the heart-sounds are clearer, often sharp, valvular, or fœtal in character; whereas in effusion the sounds are distant and muffled.

(*e*) Rarely in dilatation is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region.

The number of excellent observers who have acknowledged that they have failed sometimes to discriminate between these two conditions, and who have indeed performed paracentesis *cordis* instead of paracentesis *pericardii*, is perhaps the best comment on the difficulties which certain cases present.

Massive (1½ to 2 litre) exudations have been confounded with a pleural effusion. On more than one occasion the pericardium has been tapped under the impression that the exudate was pleuritic. The flat tympany in the infrascapular region, the absence of well-defined movable dulness, and the feeble, muffled sounds are indicative points. If the case has been followed from day to day there is rarely much difficulty; but it is different when a case presents a large area of dulness in the antero-lateral region of the left chest, and there is no to-and-fro pericardial friction murmur. Many of the cases have been regarded as encapsulated pleural effusion.

The nature of the fluid cannot positively be determined without aspiration; but a fairly accurate opinion can be formed by the nature of the primary disease and the general condition of the patient. In rheumatic cases the exudation is usually sero-fibrinous; in septic and tuberculous cases it is often purulent from the outset; in senile, nephritic, and tuberculous cases the exudation is sometimes hæmorrhagic.

Treatment.—The patient should have absolute quiet, mentally and bodily, so as to reduce to a minimum the heart's action. Drugs given for this purpose, such as aconite or digitalis, are of doubtful utility. Local bloodletting by cupping or leeches is certainly advantageous in robust subjects, particularly in the cases of extension in pleuro-pneumonia. The ice-bag ~~or Leiter's tube may be used to advantage. They have the double effect of reducing the heart's action and retarding the progress of inflammation.~~ Blisters are not indicated in the early stage.

When effusion is present, the following measures to promote absorption may be adopted: Blisters to the præcordia, a practice not so much in vogue now as formerly. It is surprising, however, in some instances, how quickly an effusion will subside on their application. If the patient's

Machine: Cobb

far

~~Applied for at intervals~~ over the heart seems to lessen the liability to effusion and my recent experience fully bears out the statements of D. Lees as to great value in this affection. It may be applied for

strength is good, a purge every other morning may be given. The diet should be light, dry, and nutritious. In cases in which the pulse is strong and the constitutional disturbance not great, iodide of potassium may be of service, and the action of the kidneys may be promoted by the infusion of digitalis and acetate of potash.

When the effusion is large, as soon as signs of serious impairment of the heart occur, as indicated by dyspnœa, small rapid pulse, dusky, anxious countenance, surgical measures should be resorted to, and paracentesis, or incision of the pericardium, at once be performed. With the sero-fibrinous exudate, such as commonly occurs after rheumatism, aspiration is sufficient; but when the exudate is purulent the pericardium should be freely incised and freely drained. The puncture may be made in the fourth interspace, either at the left sternal margin or 2·5 cm. (an inch) from it. If made in the fifth interspace it is well to puncture an inch and a half from the left sternal margin. In large effusions the pericardium can also be readily reached without danger by thrusting the needle upward and backward close to the costal margin in the left costo-xiphoid angle. The results of paracentesis of the pericardium have so far not been satisfactory. With an earlier operation in many instances and a more radical one in others—a free incision and not aspiration when the fluid is purulent—the percentage of recoveries will be greatly increased.

Chronic Adhesive Pericarditis (*Adherent Pericardium*).—This condition follows acute pericarditis, and may be partial or universal. It is not very uncommon to meet with limited synechia over the right ventricle. In the mildest grades of complete adhesion the amount of connective tissue between the membranes is slight, and there is not much thickening. These are the instances which follow the fibrinous rheumatic pericarditis. The most extreme thickening of the membranes is met with in the chronic tuberculous form, which has already been described, and which is much more common than indicated in the literature. After the absorption of an extensive purulent or sero-purulent exudate the inspissated remnants may undergo calcification. This may be in quite a limited region, most frequently over the auricles or at the base of the heart. In extreme grades the organ is completely invested by a calcareous membrane, which in places may be from 1 to 1·5 cm. in thickness.

The *symptoms* of adherent pericardium are uncertain and indefinite. A majority of the cases are met with accidentally in the post-mortem room, and there may have been no indications whatever during life of cardiac disturbance. Enlargement of the heart is an almost constant accompaniment of universal adhesion, and many of the cases come under observation for the first time with failure of this hypertrophy and signs of cardiac insufficiency.

The following are the important points in the diagnosis:

(1) *Inspection.*—In children, in whom the condition is not very un-

common as a sequence of rheumatism, the hypertrophied heart causes bulging of the chest wall. The area of cardiac impulse is increased and may sometimes be seen from the third to the sixth interspace and beyond the nipple line. The strongest impulse may be to the right of the apex. The wavy character of the pulsation in the third, fourth, and fifth interspaces is not peculiar to adherent pericardium. Not much stress can be laid upon the fixed position of the impulse, which in great enlargement of the heart is not much influenced either by posture or respiration. A more important point is systolic retraction of the apex region. Whether this occurs without adhesion of the pericardium to the chest wall is doubtful. It is often marked, and is sometimes best appreciated by the application of the hand over the apex region, which is felt to be drawn in at the moment of systole. The retraction may be most noticeable in the lower sternal region or even at the xiphoid cartilage. Following this there is sometimes a rapid rebound—the diastolic shock—which has been regarded by some as the most reliable of all signs of pericardial adhesion. Associated with this diastolic rebound is the so-called *Friedrich's sign*—diastolic collapse of the cervical veins.

(2) *Percussion* reveals an increase in the area of cardiac dulness, particularly upward as high as the second interspace. In a majority of the cases there are adhesions as well between the pleura and pericardium—in ten of thirteen cases analyzed by Ord. In some instances the dulness may reach as high as the first interspace. A sign of value is the fixed limit above and to the left of cardiac dulness, as pointed out by C. J. B. Williams. When the outer layer of the pericardium is adherent to the pleura this is a sign of very definite value, and the limit of dulness varies very slightly on deep inspiration.

(3) On *auscultation* the phenomena vary extremely with the condition of the chambers. There may be no murmurs. When extreme dilatation is present the gallop or fœtal rhythm occurs. A loud ~~regurgitant~~ murmur is not uncommon at the apex region, and the cases are frequently mistaken for mitral ~~insufficiency~~ disease.

(4) The *pulsus paradoxus* in which during inspiration the pulse-wave is small and feeble, is sometimes present, but it is not a diagnostic sign of either simple pericardial adhesion or of the cicatricial mediastino-pericarditis.

Adherent pericardium with extreme dilatation of the heart may raise the suspicion of pericarditis with effusion, as the outline of dulness in both is somewhat alike. As a rule, however, the basic dulness is broader in adhesion, and has not the pear-shaped outline. The extent and wavy character of the impulse is never so marked in large effusions, and the heart-sounds are muffled.

Machine: Cable

II. OTHER AFFECTIONS OF THE PERICARDIUM.

1. Hydropericardium.—Naturally there are in the pericardial sac a few cubic centimetres of clear, citron-colored fluid, which probably represents a post-mortem transudate. In certain conditions during life there may be large secretions of serum forming what is known as dropsy of the pericardium. It occurs usually in connection with general dropsy, due to kidney or heart disease; more commonly the former. It rarely of itself proves fatal, though when the effusion is excessive it adds to the embarrassment of the heart and the lungs, particularly when the pleural cavities are the seat of similar exudation. There are rare instances in which effusion into the pericardium occurs after scarlet fever with few, if any, other dropsical symptoms. The physical signs are those already referred to in connection with pericarditis with effusion. It is frequently overlooked.

In rare cases the serum has a milky character—chylo-pericardium.

2. Hæmo-pericardium.—This condition, by no means uncommon, is met with in aneurism of the first part of the aorta, of the cardiac wall, or of the coronary arteries, and in rupture and wounds of the heart. Death usually follows before there is time for the production of symptoms other than those of rapid heart-failure due to compression. Particularly is this the case in aneurism. In rupture of the heart the patient may live for many hours or even days with symptoms of progressive heart-failure, dyspnœa, and the physical signs of effusion.

As already mentioned, the inflammatory exudate of tubercle or cancer is often blood-stained. The same is true of the effusion in the pericarditis of Bright's disease and of old people.

3. Pneumo-pericardium.—Gas is rarely found in the pericardial sac, and is due, as a rule, to perforation from without, as in the case of stab wounds, or as the result of perforation from within, as in cancer of the stomach. Possibly, too, it may result from the decomposition of a purulent exudate. As a result of perforation, acute pericarditis is always excited, and the effusion rapidly becomes purulent. The physical signs are remarkable. When the effusion is copious the fluid and gas together give a movable area of percussion dulness with marked tympany in the region of the gas. On auscultation, remarkable splashing, churning, metallic phenomena are heard with friction and possibly feeble, distant heart-sounds. Death follows rapidly, even in thirty-six hours, as in a case (the only one which I have seen) of perforation of the pericardium in cancer of the stomach. Except as a result of injury, the condition is not one for which treatment is available. In a case of perforation from without with signs of effusion, to enlarge the wound by free incision would be justifiable.

Systolic (? Hall Denver C.T.

II. OTHER AFFECTIONS OF THE PERICARDIUM.

1. **Hydropericardium.**—Naturally there are in the pericardial sac a few cubic centimetres of clear, citron-colored fluid, which probably represents a post-mortem transudate. In certain conditions during life there may be large secretions of serum forming what is known as dropsy of the pericardium. It occurs usually in connection with general dropsy, due to kidney or heart disease; more commonly the former. It rarely of itself proves fatal, though when the effusion is excessive it adds to the embarrassment of the heart and the lungs, particularly when the pleural cavities are the seat of similar exudation. There are rare instances in which effusion into the pericardium occurs after scarlet fever with few, if any, other dropsical symptoms. The physical signs are those already referred to in connection with pericarditis with effusion. It is frequently overlooked.

In rare cases the serum has a milky character—chylo-pericardium.

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II. DISEASES OF THE HEART.

I. ENDOCARDITIS.

Inflammation of the lining membrane of the heart is usually confined to the valves, so that the term is practically synonymous with valvular endocarditis. It occurs in two forms—*acute*, characterized by the presence of vegetations with loss of continuity or of substance in the valve tissues; *chronic*, a slow sclerotic change, resulting in thickening, puckering, and deformity.

Acute Endocarditis.

This occurs in rare instances as a primary, independent affection; but in the great majority of cases it is an accident in various infective processes, so that in reality the disease does not constitute an etiological entity.

For convenience of description we speak of a simple or benign, and a malignant or ulcerative endocarditis, between which, however, there is no essential anatomical difference, as all gradations can be traced, and they represent but different degrees of intensity of the same process.

Simple Endocarditis.—This is characterized by the presence on the valves or on the lining membrane of the chambers of minute vegetations, ranging from 1 to 4 mm. in size, with an irregular and fissured surface, giving to them a warty or verrucose appearance. Often these little cauliflower-like excrescences are attached by very narrow pedicles. It is rare to see any swelling or infiltration of the endocardium in the neighborhood of even the smallest of the granulations, and although small capillary vessels do exist at the edges of the valves, redness, indicative of the injection or distention of the vessels, is extremely rare. With time the vegetations may increase greatly in size, but in what may be called simple endocarditis the size rarely exceeds that mentioned above. The finer changes in the process consist of the proliferation of the subendothelial connective-tissue elements, resulting in a small-celled infiltration. What part, if any, the endothelial cells play in this is not accurately known. The superficial elements undergo a coagulation necrosis, and fibrin is deposited from the blood, often in layers. Practically a vegetation is a small area of granulation tissue capped with fibrin. Micro-organisms are present, entangled in the granular and fibrillated fibrin, but whether they constitute an essential and constant element in all cases of simple endocarditis has not yet been decided.

The further changes in the vegetation may be either in the direction of increased proliferation of the connective-tissue elements of the valve, forming an extensive area of necrosis and the production of the condition which, from its more intense grade, we speak of as malignant or ulcerative endocarditis; or, as is more usual, healing occurs. The vegetation is

absorbed, and there remains a small nodular thickening of the valve. A third possibility is the dislocation of a vegetation with transference as an embolus to a distant part of the circulation. It is to be noted, however, that this untoward event is rare in acute endocarditis associated with febrile affections, whereas it is by no means uncommon in the simple endocarditis which occurs so constantly on old sclerotic valves.

Anatomically, in the majority of instances of acute endocarditis, cicatrization of the granulation tissue takes place in time, with but little damage to the valve beyond slight nodular thickening. The essential danger is remote and results from the slow changes in the valve tissue, which are so apt to follow an acute inflammation. Why this should be so cannot at present be explained; but the fact remains that the simple endocarditis, harmless in itself, such as we meet with in rheumatism or in chorea, lays the foundation of subsequent organic lesions, owing to the initiation of nutritive changes leading to sclerosis with contraction and deformity.

Endocarditis is much more common on the left side of the heart and involves the valvular endocardium in the great majority of cases. During fœtal life the right side of the heart is often affected. The chordæ tendineæ are sometimes involved with the valves, rarely alone. The mitral valves are more often affected than the aortic. On the mitral segment the vegetations are usually on the auricular face, not at the margin, but at a distance of 2 or 3 mm., forming a row of bead-like outgrowths. So, too, on the aortic segment they are not seen on the free margin, but just below, on the ventricular face, following the margin of the so-called lunated spaces. In both the valves this peculiar distribution follows, as Sibson suggests, the lines of maximum contact.

Etiology.—Simple endocarditis does not constitute a disease of itself, but is invariably found with some other affection. The general experience of the profession has confirmed the original observation of Bouillaud as to the frequency of association of simple endocarditis with acute articular rheumatism. Possibly it is nothing in the disease itself, but simply an altered state of the fluid media—a reduction perhaps of the lethal influences which they normally exert—permitting the invasion of the blood by certain micro-organisms. Tonsillitis, which in some forms is regarded as a rheumatic affection, may be complicated with endocarditis. Of the specific diseases of childhood it is not uncommon in scarlet fever, while it is rare in measles and chicken-pox. In diphtheria simple endocarditis is rare. It was not present in a single instance of 30 autopsies which I made in this disease at the Montreal General Hospital. In small-pox it is not common. It is stated to be more frequent in typhoid fever but was not present in 65 post-mortems in this disease.

In pneumonia both simple and malignant endocarditis are common. In 100 autopsies in this disease made at the Montreal General Hospital there were 5 instances of the former. Acute endocarditis is by no

means rare in phthisis. I have met with it in 12 cases in 216 post-mortems.

In chorea simple warty vegetations are found on the valves in a large majority of all fatal cases. There is no disease in which, post mortem, acute endocarditis has been so frequently found. And lastly, simple endocarditis is met with in diseases associated with loss of flesh and progressive debility, as cancer, and such disorders as gout, diabetes, and Bright's disease.

A very common form is that which occurs on the sclerotic valves in old heart-disease—the so-called recurring endocarditis.

Symptoms.—Neither the clinical course nor the physical signs are in any respect characteristic. The great majority of the cases are latent and there is no indication whatever of cardiac mischief. Experience has taught us that endocarditis is frequently found post mortem in persons in whom it was not suspected during life. There are certain features, however, by which its presence is indicated with a degree of probability. The patient, as a rule, does not complain of any pain or cardiac distress. In a case of acute rheumatism, for example, the symptoms to excite suspicion would be increased rapidity of the heart's action, perhaps slight irregularity, and an increase in the fever without aggravation of the joint trouble. Rows of tiny vegetations on the mitral or on the aortic segments seem a trifling matter to excite fever and it is difficult in the endocarditis of febrile processes to say definitely in every instance that an increase in the fever depends upon the endocardial complication. But a study of the recurring endocarditis—which is of the warty variety, consisting of minute beads on old sclerotic valves—shows that this process may be associated, for days or weeks at a time, with slight fever ranging from 100° to 102½°. Palpitation may be a marked feature and is a symptom upon which certain authors lay great stress.

The *diagnosis* of the condition rests upon physical signs which are notoriously uncertain. The presence of a murmur at one or other of the cardiac areas in a case of fever is often regarded as indicative of the existence of endocarditis. This extremely common mistake has arisen from the fact that the *bruit de souffle* or bellows murmur is common to endocarditis and a number of other conditions which have nothing to do with it. At first there may be only a slight roughening of the first sound, which may gradually develop into a distinct murmur. Taken alone, it is, however, a very uncertain and fallacious sign.

Malignant Endocarditis.—Acute endocarditis of a malignant character is met with:

(*a*) As a primary disease of the lining membrane of the heart or of its valves.

(*b*) As a secondary affection in acute rheumatism, pneumonia, and in various specific fevers; or as an associated condition in septic processes.

It is also known by the names of ulcerative, infectious, or diphtheritic endocarditis, but the term malignant seems most appropriate to characterize the essential clinical features of the disease.

Etiology.—The existence of a primary endocarditis has been doubted; but there are instances in which persons previously in good health, without any history of affections with which endocarditis is usually associated, have been attacked with symptoms resembling severe typhus or typhoid. In one case which I saw death occurred on the sixth day and no lesions were found other than those of malignant endocarditis.

Rheumatism, with which simple endocarditis is frequently associated, is not so often complicated with the malignant form. Thus, in only 24 of 209 cases the symptoms of severe endocarditis arose in the progress of acute or subacute rheumatism. In only 3 of the Montreal cases was there a history of rheumatism either before or during the attacks.

Malignant endocarditis is extremely rare in chorea. Of all acute diseases complicated with severe endocarditis pneumonia probably heads the list. This fact, which had been referred to by several of the older writers, was brought out in a striking manner by the figures on which my lectures were based. In 11 of the 23 Montreal cases the disease came on with lobar pneumonia, while it developed with this disease in 54 of the 209 cases analyzed—indeed, the endocarditis which occurs in pneumonia seems to be of an unusually malignant type, as in 16 cases of my 100 autopsies in this disease in which this lesion was present, 11 were of this form. Meningitis was associated with endocarditis in 25 of the 209 cases, and in 15 there was also pneumonia.

The affection may complicate erysipelas, septicæmia (from whatever cause), and puerperal fever and gonorrhœa. Malignant endocarditis is very rare in tuberculosis, typhoid fever, and diphtheria.

It has been stated by many writers that endocarditis occurs in ague. With the unusual facilities for the study of this disease which I have had in the past seven years I have not yet met with an instance. Unquestionably, in the majority of these cases, the intermittent pyrexia, which has been regarded as characteristic of the ague, has depended upon the endocarditis. In dysentery cases have been described. In small-pox and scarlet fever, with which simple endocarditis is not infrequently complicated, the malignant form is extremely rare.

Morbid Anatomy.—The lesions may be either vegetative, ulcerative, or suppurative, and these forms may occur alone or in combination. Even with vegetations there is distinct necrosis and loss of the endocardial substance. More frequently there is ulceration, either superficial, involving only the endocardium, or deep and distinct, leading to perforation of a valve, of a septum, or even of the heart itself. In the suppurative form the deeper tissues of the valve appear first affected and small abscesses are found at the bases of the vegetations. The vegetations may present a remarkable greenish-gray or greenish-yellow color, and when of long stand-

ing, or even in cases which from the clinical history appear to be tolerably acute, the vegetations may be crusted with lime salts.

A large vegetation of malignant endocarditis consists histologically of a granular and fibrillated fibrin, colonies of micro-organisms, and distinct granulation tissue at the base, while the subjacent endocardial layers show infiltration and proliferation. The destruction of tissue results from a gradual extension of the necrotic processes. Various micro-organisms have been found in connection with the disease, and the following brief statement may be made with reference to them: In a large proportion of the cases streptococci and staphylococci are found. The pneumococcus has been cultivated from the vegetations in pneumonia. Other forms have occasionally been met with.

The following figures, taken from my Gulstonian lectures at the Royal College of Physicians, give an approximate estimate of the frequency with which in 209 cases different parts of the heart were affected: Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves alone, 77; tricuspid in 19; the pulmonary valves in 15; and the heart wall in 33. In 9 instances the right heart alone was involved.

Mural endocarditis is seen most often at the upper part of the septum of the left ventricle. Next in order is the endocarditis of the left auricle on the postero-external wall. The ulcerative changes may lead to perforation of a valve segment, erosion of the chordæ tendineæ, perforation of the septum, or even of the heart itself. A common result of the ulceration is the production of valvular aneurism. In three fourths of the cases the affected valves present old sclerotic changes. The process may extend to the aorta, producing, as in one of my cases, extensive endarteritis with multiple acute aneurisms.

The associated pathological changes are partly those of the primary disease to which the endocarditis is secondary and partly those due to embolism. In the endocarditis of septic processes there is the local lesion —an acute necrosis, a suppurative wound, or puerperal disease. In many cases the lesions are those of pneumonia, rheumatism, or other febrile processes. The changes due to embolism constitute the most striking features, but it is remarkable that in some instances, even with endocarditis of a markedly ulcerative character, there may be no trace of embolic processes.

The infarcts may be few in number—only one or two, perhaps, in the spleen or kidney—or they may exist in hundreds throughout the various parts of the body. They may present the ordinary appearance of red or white infarcts of a suppurative character. They are most common in the spleen and kidneys, though they may be numerous in the brain, and in many cases are very abundant in the intestines. In right-sided endocarditis there may be infarcts in the lungs. In many of the cases there are innumerable miliary abscesses. Acute suppurative meningitis was met with in 5 of 23 of the Montreal cases, and in over ten per cent of the 209

Machine: Cobb

cases analyzed in the literature. Acute suppurative parotitis also may occur.

Symptoms.—It is difficult to give a satisfactory clinical picture of the disease because the modes of onset are so varied and the symptoms so diverse. Arising in the course of some other disease, there may be simply an intensification of the fever or a change in its character. In a majority of the cases there are present certain general features, such as irregular pyrexia, delirium, sweating, gradual failure of strength.

Embolic processes may give special characters, such as delirium, coma or paralysis from involvement of the brain or its membranes, pain in the sides and local peritonitis from infarction of the spleen, bloody urine from implication of the kidneys, impaired vision from retinal hæmorrhage, and suppuration, and even gangrene, in various parts from the distribution of the emboli.

Two special types of the disease have been recognized,—the septic or pyæmic and the typhoid. Other cases closely resemble true intermittent fever. In some the cardiac symptoms are most prominent, while in others again the main symptoms may be those of an acute affection of the cerebro-spinal system.

The *septic type* is met with usually in connection with an external wound, the puerperal process, or an acute necrosis. There are rigors, sweats, irregular fevers, and all of the signs of septic infection. The heart symptoms may be completely masked by the general condition, and attention called to them only on the occurrence of embolism. In a most remarkable sub-group of this type the disease may simulate a quotidian or a tertian ague. The symptoms may develop in persons with chronic heart-disease without any external lesions. These cases may be much prolonged—for three or four months, or even longer, as in a case of Brisbane's. The existence in some of these instances of a previous genuine malaria has been a very puzzling circumstance.

The *typhoid type* is by far the most common and is characterized by an irregular temperature, early prostration, delirium, somnolence, and coma, relaxed bowels, sweating, which may be of a most drenching character, petechial and other rashes, and occasionally parotitis. The heart symptoms may be completely overlooked, and in some instances the most careful examination has failed to discover a murmur.

Under the *cardiac group*, as suggested by Brunwell, may be considered those cases in which patients with chronic valve disease are attacked with marked fever and evidence of recent endocarditis. Many such cases present symptoms of the pyæmic and typhoid character and may run a most acute course. In others the process is less intense and the course more chronic, lasting for weeks or months, so that the term acute is scarcely applicable to them. In a case of this kind under the care of Mullin, of Hamilton, the irregular fever lasted for more than a year. The autopsy showed extensive vegetative and ulcerative disease of the mitral valves.

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There are cases in which it is often difficult to decide whether malignant endocarditis is present or not. Thus, a patient with aortic valve disease is under treatment for failing compensation and begins to have irregular fever with restlessness and cardiac distress; embolic phenomena may develop—sudden hemiplegia, pain in the region of the spleen, or bloody urine, or perhaps peripheral embolism. There may be a low delirium and the case may run a tolerably acute course; but in other instances the fever subsides and recovery occurs.

In what may be termed the *cerebral group* of cases the clinical picture may simulate a meningitis, either basilar or cerebro-spinal. There may be acute delirium or, as in three of the Montreal cases, the patient may be brought into the hospital unconscious. Heineman reports an instance, with autopsy, in which the clinical picture was that of an acute cerebro-spinal meningitis.

Certain special symptoms may be mentioned. The fever is not always of a remittent type, but may be high and continuous. Petechial rashes are very common and render the similarity very strong to certain cases of typhoid and cerebro-spinal fevers. In one case the disease was thought to be hæmorrhagic small-pox. Erythematous rashes are not uncommon. The sweating may be most profuse, even exceeding that which occurs in phthisis and ague. Diarrhœa is not necessarily associated with embolic lesions in the intestines. Jaundice has been observed and cases are on record which were mistaken for acute yellow atrophy.

The heart symptoms may be entirely latent and are not found unless a careful search be made. Even on examination there may be no murmur present. Instances are recorded by careful observers, in which the examination of the heart has been negative. Cases with chronic valve disease usually present no difficulty in diagnosis.

The course of the disease is varied, depending largely upon the nature of the primary trouble. Except in the disease grafted upon chronic valvulitis the course is rarely extended beyond five or six weeks. As already mentioned, there are instances in which the disease is prolonged for months. The most rapidly fatal case on record is described by Eberth, the duration of which was scarcely two days.

Diagnosis.—In many cases the detection of the disease is very difficult; in others, with marked embolic symptoms, it is easy. From simple endocarditis it is readily distinguished, though confusion occasionally occurs in the transitional stage, when a simple is developing into a malignant form. The constitutional symptoms are of a graver type, the fever is higher, rigors are common, and septic and typhoid symptoms develop. Perhaps a majority of the cases not associated with puerperal processes or bone disease are confounded with typhoid fever. A differential diagnosis may even be impossible, particularly when we consider that in typhoid fever infarctions and parotitis may occur. The diarrhœa and abdominal tenderness may also be present, which with the stupor and progressive

asthenia make a picture not to be distinguished from this disease. Points which may guide us are: The more abrupt onset in endocarditis, the absence of any regularity of the pyrexia in the early stage of the disease, and the cardiac pain. Oppression and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. Between pyæmia and malignant endocarditis there are practically no differential features, for the disease really constitutes an *arterial pyæmia* (Wilks). In the acute cases resembling malignant fevers, the diagnosis is usually made of typhus, typhoid, cerebro-spinal fever, or even of hæmorrhagic small-pox. The intermittent pyrexia, occurring for weeks or months, has led in some cases to the diagnosis of malaria, but this disease could now be positively excluded by the blood examination.

The cases usually terminate fatally. The instances of recovery are those more subacute forms, the so-called recurring endocarditis developing on old sclerotic valves in cases of chronic heart-disease.

Treatment.—We know no measures by which in rheumatism, chorea, or the eruptive fevers the onset of endocarditis can be prevented. As it is probable that many cases develop, particularly in children, in mild forms of these diseases, it is well to guard the patients against taking cold and insist upon rest and quiet, and to bear in mind that of all complications an acute endocarditis, though in its immediate effects harmless, is perhaps the most serious. This statement is enforced by the observations of Sibson that on a system of absolute rest the proportion of cases of rheumatism attacked by endocarditis was less than of those who were not so treated.

It is doubtful whether the salicylates in rheumatism have an influence in reducing the liability to endocarditis. When the endocarditis is present we know no remedies which will definitely influence the valvular lesions. If there is much vascular excitement aconite may be given and an ice-bag placed over the heart.

The salicylates are strongly advised by some writers and the sulphocarbolates have been recommended by Sansom. In the severer cases of malignant endocarditis the treatment is practically that of septicæmia.

Chronic Endocarditis.

This condition, which is a sclerosis of the valve, may be primary, but is oftener secondary to acute endocarditis, particularly the rheumatic form. It is essentially a slow, insidious process which leads to deformity of the valve segment and is the foundation of chronic valvular disease.

Certain poisons appear capable of initiating the change, such as alcohol, syphilis, and gout, though we are at present ignorant of the way in which they act. A very important factor, particularly in the case of the aortic valves, is the strain of prolonged and heavy muscular exertion. In no other way can be explained the occurrence of so many cases of sclero-

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sis of the aortic valves in young and middle-aged men whose occupations necessitate the overuse of the muscles.

Morbid Anatomy.—Vegetations in the form in which they occur in acute endocarditis are not present. In the early stage, which we have frequent opportunities of seeing, the edge of the valve is a little thickened and perhaps presents a few small nodular prominences, which in some cases may represent the healed vegetations of the acute process. In the aortic valves the tissue about the corpora Arantii is first affected, producing a slight thickening with an increase in the size of the nodules. The substance of the valve may lose its translucency, and the only change noticeable is a grayish opacity and a slight loss of its delicate tenuity. In the auriculo-ventricular valves these early changes are seen just within the margin and here it is not uncommon to find swellings of a grayish-red, somewhat infiltrated appearance, almost identical with the similar structures on the intima of the aorta in arterio-sclerosis. Even early there may be seen yellow or opaque-white subintimal fatty areas. As the sclerotic changes increase the fibrous tissue contracts and produces thickening and deformity of the segment, the edges of which become round, curled, and incapable of that delicate apposition necessary for perfect closure. A sigmoid valve, for instance, may be narrowed one fourth or even one third across its face, inducing the most extreme grade of insufficiency without any special deformity and without any definite narrowing of the arterial orifice. In the auriculo-ventricular segments a simple process of thickening and curling of the edges of the valves, inducing a failure to close without forming any obstruction to the normal course of the blood-flow, is less common. Still, we meet with instances at the mitral orifice, particularly in children, in which the edges of the valves are curled and thickened, producing extreme insufficiency without any material narrowing of the orifice. More frequently, as the disease advances, the chordæ tendineæ become thickened, first at the valvular ends and then along their course. The edges of the valves at their angles are gradually drawn together and there is a definite narrowing of the orifice, leading in the aorta to more or less stenosis and in the left auriculo-ventricular orifice—the two most frequently involved—to constriction. Finally, in the sclerotic and necrotic tissues lime salts are deposited and may even reach the deeper structures of the fibrous rings, and the entire valve becomes a dense calcareous mass with scarcely a remnant of normal tissue. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valve. The apices of the papillary muscles usually show marked fibroid change.

In all stages of the process the vegetations of simple endocarditis may be found and upon sclerotic valves we find the severer, ulcerative form of the disease.

Chronic *mural* endocarditis produces cicatricial-like patches of a gray-

ish-white appearance which are sometimes seen on the muscular trabeculæ of the ventricle or in the auricles. It often occurs in association with myocarditis.

The frequency with which chronic endocarditis is met with may be gathered from the following figures: In the statistics, amounting to from 12,000 to 14,000 autopsies, reported from Dresden, Wurzburg, and Prague the percentage ranged from four to nine. The relative frequency of involvement of the various valves is thus given in the collected statistics of Parrot: The mitral orifice was involved in 671, the aortic in 380, the tricuspid in 46, and the pulmonary in 11. This gives 57 instances in the right to 1,001 in the left heart.

The endocarditis of the fœtus is usually of the sclerotic form and involves the valves of the right more frequently than those of the left side.

The effects of sclerotic endocarditis are practically those of chronic valvular disease, and the general influence on the work of the heart may be briefly stated as follows: The sclerosis induces insufficiency or stenosis, which may exist separately or in combination. The narrowing retards in a measure the normal outflow and the insufficiency permits the blood current to take an abnormal course. In both instances the effect is dilatation of a chamber. The result in the former case is an increase in the difficulty which the chamber has in expelling its contents through the narrow orifice; in the other, the overfilling of a chamber by blood flowing into it from an improper source, as, for instance, in mitral insufficiency, when the left auricle receives blood both from the pulmonary veins and from the left ventricle.

The cardiac mechanism is fully prepared to meet ordinary grades of dilatation which constantly occur during sudden exertion. A man, for instance, at the end of a hundred-yard race has his right chambers greatly dilated and his reserve cardiac power worked to its full capacity. The slow progress of the sclerotic changes brings about a gradual, not an abrupt, insufficiency, and the moderate dilatation which follows is at first overcome by the exercise of the ordinary reserve strength of the heart muscles. Gradually a new factor is introduced. The reserve power which is capable of meeting sudden emergencies in such a remarkable manner is unable to cope long with a permanent and perhaps increasing dilatation. More work has to be done and, in accordance with definite physiological laws, more power is given by increase of the muscles. The heart hypertrophies and the effect of the valve lesion becomes, as we say, *compensated*. The equilibrium of the circulation is in this way maintained.

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II. CHRONIC VALVULAR DISEASE.

Aortic Incompetency.

Incompetency of the aortic valves arises either from inability of the valve segments to close an abnormally large orifice or more commonly from disease of the segments themselves. This best-defined and most easily recognized of valvular lesions was first carefully studied by Corrigan, whose name it sometimes bears.

Etiology and Morbid Anatomy.—It is more frequent in males than in females, affecting chiefly able-bodied, vigorous men at the middle period of life. The ratio which it bears to other valve diseases has been variously given from thirty to fifty per cent.

Among the important factors in producing this condition are: (*a*) Congenital malformation, particularly fusion of two segments — most commonly those behind which the coronary arteries are given off. It is probable that an aortic orifice may be competent with this bicuspid state of the valves, but a great danger is the liability of these malformed segments to sclerotic endocarditis. Of seventeen cases which I have reported all presented sclerotic changes, and the majority of them had, during life, the clinical features of chronic heart-disease.

(*b*) Acute endocarditis. This does not produce aortic incompetency unless the process passes on to ulceration and destruction, under which circumstances it is often found, and may cause a rapidly fatal issue. Simple endocarditis associated with the specific fevers is not nearly so common on the aortic as on the mitral segments; so also with rheumatism, which plays a less important *rôle* here than in mitral valve disease.

(*c*) By far the most frequent cause of insufficiency is the slow, progressive sclerosis of the segment, resulting in a curling of the edge, which lessens the working surface of the valve. This may, of course, follow acute endocarditis, but it is so often met with in strong, able-bodied men among the working classes, without any history of rheumatism or special febrile diseases with which endocarditis is commonly associated, that other conditions must be sought for to explain its frequency. Of these, unquestionably strain is the most important—not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle. Of circumstances increasing this tension, heavy and excessive use of the muscles is perhaps the most important. So often is this form of heart-disease found in persons devoted to athletics that it is sometimes called the "athlete's heart." Alcohol is a second important factor, and is stated to raise considerably the tension in the aortic system. A combination of these two causes is extremely common. A third element in inducing chronic sclerotic changes in these valves is syphilis. Cases are rarely seen in which other factors must not be taken into account, but the association is too

frequent to be accidental. That syphilis is capable of inducing arterial sclerosis is, I think, acknowledged, although the way in which it is done is not yet clear. It is interesting to note with what frequency this form of valve disease occurs in soldiers. I was struck with this fact in the Philadelphia Hospital, to which so many veterans of the civil war are admitted. I was in the habit of enforcing upon my students the etiological lesson by a mythological reference to Bacchus and Vulcan, at whose shrines a majority of the cases of aortic insufficiency have worshipped, and not a few at that of Venus.

The condition of the valves is such as has already been described in chronic endocarditis. It may be noted, however, how slight a grade of curling may produce serious incompetency. Associated with the valve disease is, in a majority of the cases, a more or less advanced arterio-sclerosis of the arch of the aorta, one serious effect of which may be a narrowing of the orifices of the coronary arteries. The sclerotic changes are often combined with atheroma, either in the fatty or calcareous stage. This may exist at the attached margin of the valves without inducing insufficiency. In other instances insufficiency may result from a calcified spike projecting from the aortic attachment into the body of the valve, and so preventing its proper closure. Some writers (Peter) have laid great stress upon the extension of the endarteritis to the valve, and would separate the instances of this kind from those of simple valvular endocarditis. I must say that I have not been able to recognize clinical differences between these two conditions, though anatomically we may separate the cases into two groups—those with and those without arterio-sclerosis.

(*d*) And, lastly, insufficiency may be induced by rupture of a segment —a very rare event in healthy valves, but not uncommon in disease, either from excessive strain during heavy lifting or from the ordinary endarterial strain in a valve eroded and weakened by ulcerative endocarditis.

Relative insufficiency of the sigmoid valves, due to dilatation of the aortic ring, is a rare condition. It is said to occur in extensive arterial sclerosis of the ascending portion of the arch with great dilatation just above the valves. I have myself never met with a pure instance of the kind, for in such cases I have always found the valve segments involved with the arterial coats. In aneurism just above the aortic ring, relative insufficiency of the valve may be present.

It would appear from the careful measurements of Beneke that the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one and twenty it is about 60 mm. At this it remains until the age of forty, beyond which date there is a gradual increase in the size up to the age of eighty, when it may reach from 68 to 70 mm. There is thus at the very period of life in which sclerosis of the valve is most common a physiological tendency toward the production of a state of relative insufficiency.

The insufficiency may be combined with various grades of narrowing, but the majority of the cases of aortic insufficiency present no signs of stenosis. On the other hand, cases of aortic stenosis almost without exception are associated with some grade, however slight, of regurgitation.

The direct effect of aortic insufficiency is the regurgitation of blood from the artery into the ventricle, causing an overdistention of the cavity and a reduction of the blood column; that is, a relative anæmia in the arterial tree. As an immediate effect of the double blood-flow into the left ventricle dilatation of the chamber occurs, and finally hypertrophy. In this way the valve defect is compensated and as with each ventricular systole a larger amount of blood is propelled into the arterial system, the regurgitation of a certain amount during diastole does not, for a time at least, seriously impair the nutrition of the peripheral parts. In this valve lesion dilatation and hypertrophy reach their most extreme limit. The heaviest hearts on record are described in connection with this affection. The so-called bovine heart, *cor bovinum*, may weigh 35 or 40 ounces, or even, as in a case of Dulles's, 48 ounces. The dilatation is usually extreme, and is in marked contrast to the condition of the chamber in cases of pure aortic stenosis. The papillary muscles may be greatly flattened. The mitral valves are usually not seriously affected, though the edges may present slight sclerosis, and there is often relative incompetency, owing to distention of the mitral ring. Dilatation and hypertrophy of the left auricle are common, and secondary enlargement of the right heart occurs in all cases of long standing. The myocardium usually presents changes, fibroid or fatty; more commonly the former in association with disease of the coronary arteries. The arch of the aorta may present extensive arterio-sclerosis and dilatation. In rare instances, usually the rheumatic cases, the intima is perfectly smooth, and the arch with its main branches not dilated. This condition may be found post mortem even when during life there have been the most characteristic signs of enlargement of the arch and of dilatation of the innominate and right carotid. I have even known the condition of aneurism to be diagnosed when post mortem no trace of dilatation or sclerosis was found, only an extreme grade of insufficiency with enormous dilatation and hypertrophy. The coronary arteries are usually involved in the sclerosis, and their orifices may be much narrowed. Although these vessels have been shown by Martin and Sedgwick to be filled during the ventricular systole, the circulation in them must be embarrassed in aortic incompetency. They must miss the effect of the blood-pressure in the sinuses of Valsalva during the elastic recoil of the arteries, which surely aids in keeping the coronary vessels full. The arteries of the body usually present more or less sclerosis consequent upon the strain which they undergo during the forcible ventricular systole.

Symptoms.—The condition is often discovered accidentally in persons who have not presented any features of cardiac disease.

Physical Signs.—*Inspection* shows a wide and forcible area of cardiac impulse with the apex beat in the sixth or seventh interspace, and perhaps as far out as the anterior axillary line. In young subjects the præcordia may bulge. On palpation a thrill, diastolic in time, is occasionally felt, but is not common. The impulse is usually strong and heaving, unless in conditions of extreme dilatation, when it is wavy and indefinite. *Percussion* shows a greater increase in the area of heart dulness than is found in any other valvular lesion. It extends chiefly downward and to the left.

On *auscultation* there is heard a murmur during diastole in the second right interspace, which is propagated with intensity toward the ensiform cartilage or down the left margin of the sternum toward the apex. In the majority of cases it is a soft, long-drawn *bruit*, and is of all cardiac murmurs the most reliable. It occurs during the time of, and is produced by, the reflux of blood from the aorta into the ventricle. In a large proportion of the cases there is also a systolic murmur heard at the aortic region, usually shorter, often rougher in quality, and which may be propagated upward into the neck. A common mistake is to regard this as indicating stenosis, whereas in the great majority of instances of aortic insufficiency there is no material narrowing, and the murmur is produced by roughening of the segments or of the intima of the arch. The second sound is usually obliterated, though in some instances both the murmur and the valvular sound may be distinctly heard. At the apex murmurs are also heard, either transmitted from the aortic orifice or produced at the mitral. In the majority of cases with aortic incompetency of high grade, the mitral orifice is dilated, and there is relative insufficiency of the valves. It can frequently be determined that the systolic murmur at the apex differs in quality from that at the base. A second murmur at the apex, probably produced at the mitral orifice, is not infrequent. Attention was called to this by the late Austin Flint, and the murmur usually goes by his name. It has a distinctly rumbling quality, is limited in area, and is sometimes, though not always, distinctly presystolic in time. The explanation of its occurrence, as given by Flint, is that in the extreme dilatation of the ventricle the mitral segments cannot during diastole be forced back against the wall, and, therefore, remaining in the blood current, they produce a sort of relative narrowing, and in consequence a vibratory murmur not unlike in quality the presystolic murmur of mitral stenosis. My experience as to the frequency of this murmur coincides with that of Lee.*

The examination of the arteries in aortic insufficiency is of great value. Visible pulsation is more commonly seen in the peripheral vessels in this than in any other condition. The carotids may be seen to throb forcibly, the temporals to dilate, and the brachials and radials to expand with each

* American Journal of the Medical Sciences, 1890.

heart-beat. With the ophthalmoscope the retinal arteries are seen to pulsate. Not only is the pulsation evident, but the characteristic jerking quality is apparent. In the throat the throbbing carotids may lead to the diagnosis of aneurism. In many cases the pulsation can be seen in the suprasternal notch, and prominent, forcibly-throbbing vessels beneath the right sterno-mastoid muscle. The abdominal aorta may lift the epigastrium with each systole. To be mentioned with this is the capillary pulse, met very often in aortic insufficiency, and best seen in the finger-nails or by drawing a line upon the forehead, when the margin of hyperæmia on either side alternately blushes and pales. In extreme grades the face or the hand may blush visibly at each systole. It is met with also in profound anæmia, occasionally in neurasthenia, and in health in conditions of great relaxation of the peripheral arteries. Pulsation may also be present in the peripheral veins. On palpation the characteristic water-hammer or Corrigan pulse is felt. In the majority of instances the pulse wave strikes the finger forcibly with a quick jerking impulse, and immediately recedes or collapses. The characters of this are sometimes best appreciated by grasping the arm above the wrist and holding it up. On auscultation a double murmur may be heard in the carotids and subclavians when it is present at the aortic orifice. Occasionally in the carotid the second sound is distinctly audible when absent at the aortic cartilage. In the femoral artery a double murmur also may be heard sometimes, as pointed out by Duroziez.

Aortic insufficiency may for years be fully compensated. Persons do not necessarily suffer any inconvenience, and the condition is often found accidentally. So long as the hypertrophy just equalizes the valvular defect there may be no symptoms and the individual may even take moderately heavy exercise without experiencing sensations of distress about the heart. The cases which last the longest are those in which the sclerosis follows endocarditis and is not a part of a general arterio-sclerosis. Coexistent lesions of the mitral valves tend early to disturb the compensation. It has scarcely been sufficiently recognized by the profession at large that pure aortic insufficiency is consistent with years of average health and with a tolerably active life. I know several physicians with aortic insufficiency who have been able to carry on for years large and somewhat onerous practices. One of them since the establishment of insufficiency has passed successfully through two attacks of acute rheumatism. In large hospital practice, scarcely a month passes without the discovery of a case of aortic insufficiency in connection with some other affection.

With the onset of myocardial changes, with increasing degeneration of the arteries, particularly with a progressive sclerosis of the arch and involvement of the orifices of the coronary arteries, the compensation becomes disturbed. In advanced cases the changes about the aortic ring may be associated with alterations in the cardiac nerves and ganglia, and so introduce an important factor.

Headache, dizziness, flashes of light, and a feeling of faintness on rising quickly are among the earliest symptoms. Palpitation and cardiac distress on slight exertion are common. Long before any signs of failing compensation pain may become a marked and troublesome feature. It is extremely variable in its manifestations. It may be of a dull, aching character confined to the præcordia. More frequently, however, it is sharp and radiating, and is transmitted up the neck and down the arms, particularly the left. Attacks of true angina pectoris are more frequent in this than in any other valvular disease. Anæmia is also common, much more so than in aortic stenosis or in mitral affections.

More serious symptoms, as compensation fails, are shortness of breath and œdema of the feet. The attacks of dyspnœa are liable to come on at night and the patient has to sleep with the head high or even in a chair. Of respiratory symptoms cough may develop, due to the congestion of the lungs or œdema. Hæmoptysis is less frequent than in mitral disease. I have reported a case in which it was profuse and believed to be due to tuberculosis of the lungs, inasmuch as the patient was admitted in a state of emaciation and profound exhaustion. General dropsy is not common, but œdema of the feet may occur early and is sometimes due to the anæmia, at others to the venous stasis, at times to both. Unless there is coexisting disease of the mitral valve, it is rare in pure aortic incompetency for the patient to die with general anasarca. Sudden death is frequent; more so in this than in other valvular diseases. As compensation fails the patient takes to bed and slight irregular fever, associated usually with a recurring endocarditis, is not uncommon toward the close. Embolic symptoms are not infrequent—pain in the splenic region with enlargement of the organ, hæmaturia, and in some cases paralysis. Distressing dreams and disturbed sleep are more common in this than in other forms of valvular disease.

Here may appropriately be mentioned the connection between mental symptoms and cardiac disease, as they are oftenest seen with this lesion. An admirable account of the relations between insanity and disease of the heart is to be found in Mickle's Gulstonian lectures for 1888. In general medical practice we seldom find marked mental symptoms, except toward the close of the disease, when there may be delirium, hallucinations, and morbid impulses. It is to be remembered that in many heart cases this terminal delirium is uræmic. The irritability and peevishness sometimes found in persons the subject of organic heart-disease cannot, I think, be associated with it in any special manner. We do meet insanity, breaking out in patients with aortic and mitral disease, in the stage of compensation, which appears to be related definitely to the cardiac lesion. It is important to bear this in mind, for cases occasionally display suicidal tendencies. I have twice had patients throw themselves from the window of the ward.

Aortic Stenosis.

Narrowing or stricture of the aortic orifice is not nearly so common as insufficiency. The two conditions, as already stated, may occur together, however, and probably in almost every case of stenosis there is some leakage.

Etiology and Morbid Anatomy.—In the milder grades there is adhesion between the segments, which are so stiffened that during systole they cannot be pressed back against the aortic wall. The process of cohesion between the segments may go on without great thickening, and produce a condition in which the orifice is guarded by a comparatively thin membrane, on the aortic face of which may be seen the primitive raphés separating the sinuses of Valsalva. In some instances this membrane is so thin and presents so few traces of atheromatous or sclerotic changes that the condition looks as if it had originated during fœtal life. More commonly the valve segments are thickened and rigid, and have a cartilaginous hardness. In advanced cases they may be represented by stiff, calcified masses obstructing the orifice, through which a circular or slit-like passage can be seen. The older the patient the more likely it is that the valves will be rigid and calcified.

We may speak of a relative stenosis of the aortic orifice when with normal valves and ring the aorta immediately beyond is greatly dilated. A stenosis due to involvement of the aortic ring in sclerotic and calcareous changes without lesion of the valves is referred to by some authors. I have never met with an instance of this kind. A subvalvular stenosis, the result of endocarditis in the mitro-sigmoidean sinus, usually occurs as the result of a fœtal endocarditis. In comparison with aortic insufficiency, stenosis is a rare disease. It is usually met with at a more advanced period of life than insufficiency, and the most typical cases of it are found associated with extensive calcareous changes in the arterial system in old men.

When gradually produced and when there is not much insufficiency the dilatation of the left ventricle may be slight, though I think that in all cases it does occur. The walls of the ventricle become hypertrophied, and we see in this condition the most typical instances of what is called concentric hypertrophy, in which, without much, if any, enlargement of the cavity, the walls are greatly thickened, in contradistinction to the so-called eccentric hypertrophy, in which the chamber is greatly dilated as well as hypertrophied. There may be no changes in the other cardiac cavities if compensation is well maintained; but with its failure come dilatation, impeded auricular discharge, pulmonary congestion, and increased work for the right heart. The arterial changes are, as a rule, not so marked as in aortic insufficiency, for the walls have not to withstand the impulse of a greatly increased blood-wave with each systole. On the contrary, the amount of blood propelled through the narrow orifice may be smaller than normal, though when compensation is fully established the pulse-wave may be of medium volume.

Symptoms. Physical Signs.—*Inspection* may fail to reveal any area of cardiac impulse. Particularly is this the case in old men with rigid chest walls and large emphysematous lungs. Under these circumstances there may be a high grade of hypertrophy without any visible impulse. Even when the apex beat is visible it may be, as Traube pointed out, feeble and indefinite. In many cases the apex is seen displaced downward and outward, and the impulse looks strong and forcible.

Palpation reveals in many cases a thrill at the base of the heart of maximum force in the aortic region. With no other condition do we meet with thrills of greater intensity. The apex beat may not be palpable under the conditions above mentioned, or there may be a slow, heaving, forcible impulse.

Percussion never gives the same wide area of dulness as in aortic insufficiency. The extent of it depends largely on the state of the lungs, whether emphysematous or not.

Auscultation.—A systolic murmur of maximum intensity at the aortic cartilage, and propagated into the great vessels, is present in aortic stenosis, but is by no means pathognomonic. One of the last lessons learned by the student of physical diagnosis is to recognize the fact that this systolic murmur is only in comparatively rare cases produced by decided narrowing of the aortic orifice. Roughening of the valves, or the intima of the aorta, and hæmic states are much more frequent causes. In aortic stenosis the murmur often has a much harsher quality, is louder, and is more frequently musical than in the conditions just mentioned. When compensation fails and the ventricle is dilated and feeble the murmur may be soft and distant. The second sound is rarely heard at the aortic cartilage, owing to the thickening and stiffness of the valve. A diastolic murmur is not uncommon, but in many cases it cannot be heard. The pulse in pure aortic stenosis is small, usually of good tension, regular, and perhaps slower than normal.

The condition may be latent for an indefinite period, as long as the hypertrophy is maintained. Early symptoms are those due to defective blood-supply to the brain, dizziness, and fainting. Palpitation, pain about the heart, and anginal symptoms are not so marked as in insufficiency. With degeneration of the heart-muscle and dilatation relative insufficiency of the mitral valve is established, and the patient may present all the features of engorgement in the lesser and systemic circulations, with dyspnœa, cough, rusty expectoration, and the signs of anasarca in the lower part of the body. Many of the cases in old people, without presenting any dropsy, have symptoms pointing rather to general arterial disease. Cheyne-Stokes breathing is not uncommon with or without signs of uræmia.

Diagnosis.—With an intensely rough or musical murmur of maximum intensity at the aortic region and signs of hypertrophy of the left ventricle, a thrill and a hard, slow pulse of moderate volume and fairly good tension,

a diagnosis of aortic stenosis can be made with some degree of probability, particularly if the subject is an old man. Mistakes are common, however, and a roughened or calcified valve segment, or, in some instances, a very roughened and prominent calcified plate in the aorta, and hypertrophy associated with renal disease, may produce similar symptoms.

Let me repeat that a murmur of maximum intensity at the aortic cartilage is of no importance in itself as a diagnostic sign of stenosis. Roughening of the valve, sclerosis of the intima of the arch, and anæmia are conditions more frequently associated with a systolic murmur in this region. Seldom is there difficulty in distinguishing the murmur due to anæmia, since it is rarely so intense and is not associated with thrill or with marked hypertrophy of the left ventricle. In aortic insufficiency a systolic murmur is usually present, but has neither the intensity nor the musical quality, nor is it accompanied with a thrill. With roughening and dilatation of the ascending aorta the murmur may be very harsh or musical; but the existence of a second sound, accentuated and ringing in quality, is usually sufficient to differentiate this condition.

Mitral Incompetency.

Etiology. — Insufficiency of the mitral valve results from: (*a*) Changes in the segments whereby they are contracted and shortened, usually combined with changes in the chordæ tendineæ, or with more or less narrowing of the orifice. (*b*) As a result of changes in the muscular walls of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or changes in the muscular substance, so that the segments are imperfectly coapted during the systole—muscular incompetency. The common lesions producing insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordæ tendineæ, and union of the edges of the segments, so that in a majority of the instances there is not only insufficiency, but some grade of narrowing as well. Except in children, we rarely see the mitral leaflets curled and puckered without narrowing of the orifice. Calcareous plates at the base of the valve may prevent perfect closure of one of the segments. In long-standing cases the entire mitral structures are converted into a firm calcareous ring. From this valvular insufficiency the other condition of muscular incompetency must be carefully distinguished. It is met with in all conditions of extreme dilatation of the left ventricle, and also in weakening of the muscles in prolonged fevers and in anæmia.

Morbid Anatomy.—The effects of incompetency of the mitral segment upon the heart and circulation are as follows: (*a*) The imperfect closure allows a certain amount of blood to regurgitate from the ventricle into the auricle, so that at the end of auricular diastole this chamber con-

tains not only the blood which it has received from the lungs, but also that which has regurgitated from the left ventricle. This necessitates dilatation, and, as increased work is thrown upon it in expelling the augmented contents, hypertrophy as well.

(*b*) With each systole of the left auricle a larger volume of blood is forced into the left ventricle, which also dilates and subsequently becomes hypertrophied.

(*c*) During the diastole of the left auricle, as blood is regurgitated into it from the left ventricle, the pulmonary veins are less readily emptied. In consequence the right ventricle expels its contents less freely, and in turn becomes dilated and hypertrophied.

(*d*) Finally, the right auricle also is involved, its chamber is enlarged, and its walls are increased in thickness.

(*e*) The effect upon the pulmonary vessels is to produce dilatation both of the arteries and veins—often in long-standing cases atheromatous changes; the capillaries are distended, and ultimately the condition of brown induration is produced. Perfect compensation may be effected, chiefly through the hypertrophy of both ventricles, and the effect upon the peripheral circulation may not be manifested for years, as a normal volume of blood is discharged from the left heart at each systole. The time comes, however, when, owing either to increase in the grade of the incompetency or to failure of the compensation, the left ventricle is unable to send out its normal volume into the aorta. Then there is overfilling of the left auricle, engorgement in the lesser circulation, embarrassed action of the right heart, and congestion in the systemic veins. For years this somewhat congested condition may be limited to the lesser circulation, but finally the right auricle becomes dilated, the tricuspid valves incompetent, and the systemic veins are engorged. This gradually leads to the condition of cyanotic induration in the viscera and, when extreme, to dropsical effusion.

Muscular incompetency, due to impaired nutrition of the mitral and papillary muscles, is rarely followed by such perfect compensation. There may be in acute destruction of the aortic segments an acute dilatation of the left ventricle with relative incompetency of the mitral segments, great dilatation of the left auricle, and intense engorgement of the lungs, under which circumstances profuse hæmorrhage may result. In these cases there is little chance for the establishment of compensation. In cases of hypertrophy and dilatation of the heart, without valvular lesions, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme and lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy, which cannot be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic Bright's disease the hypertrophy of the left ventricle may gradually fail, leading, in the later stages, to relative insufficiency of the mitral valve, and the production of a con-

dition of pulmonary and systemic congestion, similar to that induced by the most extreme grade of lesion of the valve itself.

Symptoms.—During the development of the lesion, unless the incompetency comes on acutely in consequence of rupture of the valve segment or of ulceration, the compensatory changes go hand in hand with the defect, and there are no subjective symptoms. So, also, in the stage of perfect compensation, there may be the most extreme grade of mitral insufficiency with enormous hypertrophy of the heart, yet the patient may not be aware of the existence of heart trouble, and may suffer no inconvenience except perhaps a little shortness of breath on exertion or on going up-stairs. It is only when from any cause the compensation has not been perfectly effected, or having been so is broken abruptly or gradually, that the patients begin to be troubled. The symptoms may be divided into two groups:

(*a*) The minor manifestations while compensation is still good. Patients with extreme incompetency often have a congested appearance of the face, the lips and ears have a bluish tint, and the venules on the cheeks may be enlarged, which in many cases is very suggestive. In long-standing cases, particularly in children, the fingers may be clubbed, and there is shortness of breath on exertion. This is one of the most constant features in mitral insufficiency, and may exist for years, even when the compensation is perfect. Owing to the somewhat congested condition of the lungs these patients have a tendency to attacks of bronchitis or hæmoptysis. There may also be palpitation of the heart. As a rule, however, in well-balanced lesions in adults, this period of full compensation or latent stage is not associated with symptoms which call the attention of the patient to an affection of the heart.

(*b*) Sooner or later comes a period of disturbed or broken compensation, in which the most intense symptoms are those of venous engorgement. There are palpitation, weak, irregular action of the heart, and signs of dilatation. Dyspnœa is a marked feature, and there may be cough. There is usually a slight cyanosis, and even a jaundiced tint to the skin. The most marked symptoms, however, are those of venous stasis. The overfilling of the pulmonary vessels accounts in part for the dyspnœa. There is cough, often with bloody or watery expectoration, and the alveolar epithelium containing brown pigment-grains is abundant. Dropsical effusion usually sets in, beginning in the feet and extending to the body and the serous sacs. The liver is enlarged, and there are signs of portal congestion, gastric irritation, and catarrh of the stomach and intestines. The urine is usually scanty and albuminous, and contains tube casts and sometimes blood-corpuscles. With judicious treatment the compensation may be restored and all the serious symptoms may pass away. Patients may have recurring attacks of this kind, but ultimately the condition is beyond repair and the patient either dies of a general

The cardiac ~~Elect~~ start, often on awaking just
as the patient falls asleep he awakes with a feeling
as if the heart was stopping and gasps for breath

dropsy or there is progressive dilatation of the heart, and death from asystole. Sudden death in these cases is rare.

Physical Signs.—*Inspection.*—In children the præcordia may bulge and there may be a large area of visible pulsation. The apex beat is to the left of the nipple, in some cases in the sixth interspace, in the anterior axillary line. There may be a wavy impulse in the cervical veins which are often full, particularly when the patient is recumbent.

Palpation.—A thrill is rare; when present it is felt at the apex, often in a limited area. The force of the impulse may depend largely upon the stage in which the case is examined. In full compensation it is forcible and heaving; when the compensation is disturbed, usually wavy and feeble.

Percussion.—The dulness is increased, particularly in a lateral direction. There is no disease of the valves which produces, in long-standing cases, a more extensive transverse area of heart dulness. It does not extend so much upward along the left margin of the sternum as beyond the right margin and to the left of the nipple line.

Auscultation.—At the apex there is a systolic murmur which wholly or partly obliterates the first sound. It is loudest here, and has a blowing, sometimes musical character, particularly toward the latter part. The murmur is transmitted to the axilla and may be heard at the back, in some instances over the entire chest. There are cases in which, as pointed out by Naunyn, the murmur is heard best along the left border of the sternum. Usually in diastole at the apex the loudly transmitted second sound may be heard. Occasionally there is also a soft, sometimes a rough or rumbling presystolic murmur. As a rule, in cases of extreme mitral insufficiency from valvular lesion with great hypertrophy of both ventricles, there is heard only a loud blowing murmur during systole. A murmur of mitral insufficiency may vary a great deal according to the position of the patient. It may be present in the recumbent and absent in the erect posture. In cases of dilatation, particularly when dropsy is present, there may be heard at the ensiform cartilage and in the lower sternal region a soft systolic murmur due to tricuspid regurgitation. An important sign on auscultation is the accentuated pulmonary second sound. This is heard to the left of the sternum in the second interspace, or over the third left costal cartilage.

The pulse in mitral insufficiency, during the period of full compensation, may be full and regular, often of low tension. Usually with the first onset of the symptoms the pulse becomes irregular, a feature which then dominates the case throughout. There may be no two beats of equal force or volume. Often after the disappearance of the symptoms of failure of compensation the irregularity of the pulse persists.

The three important physical signs then of mitral regurgitation are: (*a*) systolic murmur of maximum intensity at the apex, which is propagated to the axilla and heard at the angle of the scapula; (*b*) accentuation

of the pulmonary second sound; (*c*) evidence of enlargement of the heart, particularly the increase in the transverse diameter, due to hypertrophy of both right and left ventricles.

Diagnosis.—There is rarely any difficulty in the diagnosis of mitral insufficiency. The physical signs just referred to are quite characteristic and distinctive. Two points are to be borne in mind. First, a murmur, systolic in character, and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate incompetency of the mitral valve. There is heard in this region a large group of what are termed accidental murmurs, the precise nature of which is still doubtful. They are probably formed, however, in the ventricle, and are not associated with hypertrophy, or accentuation of pulmonary second sound.

Second, it is not always possible to say whether the insufficiency is due to lesion of the valve segment or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary second sound, nor the hypertrophy assists in the differentiation. The history is sometimes of greater value in this matter than the physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart (in which the systolic murmur may be of the greatest intensity), and the instances of arterio-sclerosis with dilated heart.

Mitral Stenosis.

Etiology.—Narrowing of the mitral orifice is usually the result of valvular endocarditis occurring in the earlier years of life; very rarely it is congenital. It is very much more common in women than in men—in 63 of 80 cases noted by Duckworth. This is not easy to explain, but there are at least two factors to be considered. Rheumatism prevails more in girls than in boys and, as is well known, endocarditis of the mitral valve is more common in rheumatism. Chorea, also, as suggested by Barlow, has an important influence, occurring more frequently in girls and often associated with endocarditis. Of 110 cases of chorea which I examined at a period more than two years subsequent to the attack, 54 cases had signs of organic heart-disease, among which were 17 instances with the physical signs of mitral stenosis. Anæmia and chlorosis, which are prevalent in girls, have been regarded as possible factors. In a number of cases, however, no recognizable etiological factor can be discovered. This has been regarded by some writers as favoring the view that many of the cases are of congenital origin; but it is not improbable that with any of the febrile affections of childhood endocarditis may be associated. Whooping-cough, too, with its terrible strain on the heart-valves, may be accountable for certain cases. Congenital affections of the mitral valve are notoriously rare. While met with at all ages, stenosis is certainly more frequent in young persons.

Morbid Anatomy.—In a majority of instances with the stenosis there is some incompetency. The narrowing results from thickening and contraction of the tissues of the ring, of the valve segments, and of the chordæ tendineæ. The condition varies a good deal according to the amount of atheromatous change. In many cases the curtains are so welded together and the whole valvular region so thickened that the orifice is reduced to a mere chink—Corrigan's button-hole contraction. In other cases the curtains are not much thickened, but narrowing has resulted from gradual adhesion at the edges, and thickening of the chordæ tendineæ, so that from the auricle it looks cone-like—the so-called funnel-shaped variety of stenosis. The instances in which the valve segments are very slightly deformed but in which the orifice is considerably narrowed, are regarded by some as possibly of congenital origin. Occasionally the curtains are in great part free from disease, but the narrowing results from large calcareous masses, which project into them from the ring. The involvement of the chordæ tendineæ is usually extreme, and the papillary muscles may be inserted directly upon the valve. In moderate grades of constriction the orifice will admit the tip of the index-finger; in more extreme forms, the tip of the little finger; and occasionally one meets with a specimen in which the orifice seems almost obliterated, as in a case which came under my notice, which only admitted a medium-sized Bowman's probe.

The heart in mitral stenosis is not greatly enlarged, rarely weighing more than 14 or 15 ounces. Occasionally, in an elderly person, it may seem slightly if at all enlarged, and again there are instances in which the weight may reach as much as 20 ounces. The left ventricle is usually small, and may look very small in comparison with the right ventricle, which forms the greater portion of the apex. In cases in which with the narrowing there is very considerable incompetency the left ventricle may be moderately dilated and hypertrophied.

These changes gradually induced are associated with secondary alterations of great importance in the heart. The left auricle discharges its blood with greater difficulty and in consequence dilates, and its walls reach three or four times their normal thickness. Although the auricle is by structure unfitted to compensate an extreme lesion, the probability is that for some time during the gradual production of stenosis, the increasing muscular power of the walls is sufficient to counterbalance the defect. Eventually the tension is increased in the pulmonary circulation, owing to impeded outflow from the veins. To overcome this the right ventricle undergoes dilatation and hypertrophy, and upon this chamber falls the work of equalizing the circulation. Relative incompetency of the tricuspid and congestion of systemic veins at last supervene.

It is not uncommon at the examination to find white thrombi in the appendix of the left auricle. Occasionally a large part of the auricle is occupied by an ante-mortem thrombus. Still more rarely the remarkable

ball thrombus is found, in which a globular concretion, varying in size from a walnut to a small egg, lies free in the auricle, two examples of which have come under my observation.

Symptoms.—Physical Signs.—*Inspection.*—In children the lower sternum and the fifth and sixth left costal cartilages are often prominent, owing to hypertrophy of the right ventricle. The apex beat may be ill-defined. Usually, it is not dislocated far beyond the nipple line, and the chief impulse is over the lower sternum and adjacent costal cartilages. Often in thin-chested persons there is pulsation in the third and fourth left interspaces close to the sternum. When compensation fails, the præcordial impulse is much feebler, and in the veins of the neck there may be marked systolic regurgitation.

Palpation reveals in a majority of the cases a characteristic, well-defined fremitus or thrill, which is best felt, as a rule, in the fourth or fifth interspace within the nipple line. It is of a rough, grating quality, often peculiarly limited in area, most marked during expiration, and can be felt to terminate in a sharp, sudden shock, synchronous with the impulse. This most characteristic of physical signs is pathognomonic of narrowing of the mitral orifice, and is perhaps the only instance in which the diagnosis of a valvular lesion can be made by palpation alone. The cardiac impulse is felt most forcibly in the lower sternum and in the fourth and fifth left interspaces. The impulse is felt very high in the third and fourth interspaces, or in rare cases even in the second, and it has been thought that in the latter interspace the impulse is due to pulsation of the auricle. It is always the impulse of the right ventricle; even in the most extreme grades of mitral stenosis, there is never such tilting forward of the auricle or its appendix as would enable it to produce an impression on the chest wall.

Percussion gives an increase in the cardiac dulness to the right of the sternum and along the left margin; not usually a great increase beyond the nipple line, except in extreme cases, when the transverse dulness may reach from 5 cm. beyond the right margin of the sternum to 10 cm. beyond the nipple line.

Auscultation.—In the mitral area, usually to the inner side of the apex beat and often in a very limited region, is heard a rough, vibratory or purring murmur, which terminates abruptly in the first sound. By combining palpation and auscultation the purring murmur is found to be synchronous with the thrill and the loud shock with the first sound. This is the presystolic murmur, about the time and mode of production of which so much discussion has occurred. I hold with those who regard it as occurring during the auricular systole. In whatever way produced, it remains one of the most distinctive and characteristic of murmurs and its presence is positively indicative of narrowing of the mitral orifice. The sole exception to this statement is the Flint murmur already referred to in aortic incompetency. Once, in a case of enormous enlargement of the

The transmission is slight, but it may sometimes be heard high in the axilla or even over the scapula (Griffith)

spleen, with dropsy, in which the heart was greatly pushed up, I heard a presystolic murmur of rough quality, and the mitral valves were found post mortem to be normal. The presystolic murmur may occupy the entire period of the diastole; but more commonly it is only the latter half, corresponding to the auricular systole. The difference may sometimes be noted between the first and second portions of the murmur, when it occupies the entire time. Often there is a peculiar rumbling or echoing quality, which in some instances is very limited and may be heard only over a single bell-space of the stethoscope. A systolic murmur may be heard at the apex or along the left sternal border, often of extreme softness and audible only when the breath is held. Sometimes the systolic murmur is loud and distinct and is transmitted to the axilla. The second sound in the second left interspace is loudly accentuated, sometimes reduplicated. It may be transmitted far to the left and be heard with great clearness beyond the apex. In uncomplicated cases of mitral stenosis there are usually no murmurs audible at the aortic region, at which spot the second sound is less intense than at the pulmonary area. In the lower sternum and to the right a tricuspid murmur is sometimes heard in advanced cases. Other points to be noted are the following: The unusually sharp, clear first sound which follows the presystolic murmur, the cause of which is by no means easy to explain. It can scarcely be a valvular sound produced chiefly at the mitral orifice, since it may be heard with great intensity in cases in which the valves are rigid and calcified. More probably it is a modified sound produced by the heart-muscle and connected, as has been suggested, with the altered conditions of the chordæ tendineæ and papillary muscles, the normal action of which must be interfered with.

These physical signs, it is to be borne in mind, are characteristic only of the stage in which compensation is maintained. Finally there comes a period in which, with rupture of compensation, the presystolic murmur disappears and there is heard in the apex region a sharp first sound, or sometimes a gallop rhythm. The marked systolic shock may be present after the disappearance of the thrill and the characteristic murmur. Under treatment, with gradual recovery of compensation, probably with increasing vigor of contraction of the right ventricle and left auricle, the presystolic murmur reappears. In cases seen at this stage of the disease the nature of the valve lesion may be entirely overlooked.

Stenosis of the mitral valve may for years be efficiently compensated by the hypertrophy of the right ventricle. Many persons with the characteristic physical signs of this lesion present no symptoms. They may for years perhaps be short of breath on going up-stairs, but are able to pass through the ordinary duties of life without discomfort. The pulse is smaller in volume than normal, but may be perfectly regular. A special danger of this stage is the recurring endocarditis. Vegetations may be whipped off into the circulation and, blocking a cerebral vessel, may cause hemiplegia or aphasia, or both. This, unfortunately, is not an uncommon

sequence in women. Patients with mitral stenosis may survive this accident for an indefinite period. A woman, over seventy years of age, died in one of my wards at the Philadelphia Hospital, who had been in the almshouse, hemiplegic, for more than thirty years. The heart presented an extreme grade of mitral stenosis which had probably existed at the time of the hemiplegic attack.

Failure of compensation brings in its train the group of symptoms which have been discussed under mitral insufficiency. Briefly enumerated they are: Rapid and irregular action of the heart, shortness of breath, cough, signs of pulmonary engorgement, and very frequently hæmoptysis. Attacks of this kind may recur for years. Bronchitis or a febrile attack may cause shortness of breath or slight blueness. Inflammatory affections of the lungs or pleura seriously disturb the right heart, and these patients stand pneumonia very badly. Many, perhaps a majority of cases of mitral stenosis, do not have dropsy. The liver may be greatly enlarged, and in the late stages ascites is not uncommon, particularly in children. General anasarca is most frequently met with in those cases in which there is secondary narrowing of the tricuspid orifice (Broadbent).

Tricuspid-Valve Disease.

(*a*) **Tricuspid Regurgitation.**—Occasionally this results from acute or chronic endocarditis with puckering; more commonly the condition is one of relative insufficiency, and is secondary to lesions of the valves on the left side, particularly of the mitral. It is met with also in all conditions of the lungs which cause obstruction to the circulation, such as cirrhosis and emphysema, particularly in combination with chronic bronchitis. The symptoms are those of obstruction in the lesser circulation with venous congestion in the systemic veins, such as has already been described in connection with mitral insufficiency. The signs of this condition are:

(1) Systolic regurgitation of the blood into the right auricle and the transmission of the pulse-wave into the veins of the neck. If the regurgitation is slight or the contraction of the ventricle is feeble there may be no venous pulsation, but in other cases there is marked systolic pulsation in the cervical veins. That in the right jugular is more forcible than that in the left. It may be seen both in the internal and the external, particularly in the latter. Marked pulsation in these veins occurs only when the valves guarding them become incompetent. Slight oscillations are by no means uncommon, even when the valves are intact. The distention of the veins is sometimes enormous, particularly in the act of coughing, when the right jugular at the root of the neck may stand out, forming an extraordinarily prominent ovoid mass. Occasionally the regurgitant pulse-wave may be widely transmitted and be seen in the subclavian and axillary veins, and even in the subcutaneous veins over the

Machine: Cobb

shoulder, or, as in a case recently under observation, in the superficial mammary veins.

Regurgitant pulsation through the tricuspid orifice may be transmitted to the inferior cava, and so to the hepatic veins, causing a systolic distention of the liver. This is best appreciated by bimanual palpation, placing one hand over the fifth and sixth costal cartilages and the other in the lateral region of the liver in the mid-axillary line. The rhythmical expansile pulsation may be readily distinguished, as a rule, from the systolic depression of the liver due to communicated pulsation from the left ventricle.

(2) The second important symptom of tricuspid regurgitation is the occurrence of a systolic murmur of maximum intensity in the lower sternum. It is usually a soft, low murmur, often to be distinguished from a coexisting mitral murmur by differences in quality and pitch, and may be heard to the right as far as the axilla. Sometimes it is very limited in its distribution.

Together these two signs positively indicate tricuspid regurgitation. In addition, the percussion usually shows increase in the area of dulness to the right of the sternum, and the impulse in the lower sternal region is forcible. In the great majority of cases the symptoms are those of the associated lesions. In cirrhosis of the lung and in chronic emphysema the failure of compensation of the right ventricle with insufficiency of the tricuspid not infrequently leads either to acute asystole or to gradual failure with cardiac dropsy.

(*b*) **Tricuspid Stenosis.**—This interesting condition may be either congenital or acquired. The congenital cases are not uncommon, and are associated usually with other valvular defects which cause early death. The acquired form is not very infrequent. Bedford Fenwick collected 46 observations, of which 41 were in women. Leudet* has analyzed 117 cases. Of 101 of these in which the ages were mentioned, 80 were in women and 21 in men. A great majority of the cases were in adults, only eight being between the ages of ten and twenty. Its rarity as an isolated condition may be gathered from the fact that of 114 autopsies, in 11 only was the lesion confined to this valve. In 21 the tricuspid, mitral, and aortic segments were involved, and in 78 the tricuspid and mitral. Practically the condition is almost always secondary to lesions of the left heart.

The physical signs are sometimes characteristic. For instance, a presystolic thrill has been noted by several observers. The percussion shows dulness to be increased, particularly to the right of the sternum. On auscultation a presystolic murmur has been determined in certain cases, and is heard best at the root of the ensiform cartilage, or a little to the right of it. Of general symptoms, cyanosis of the face and lips is very common, and in the late stages, when dropsy supervenes, it is apt to be intense.

* Paris Thesis, 1888.

The lesion is interesting chiefly because it forms one of the most serious complications of mitral stenosis.

Pulmonary Valve Disease.

This is extremely rare.

(*a*) *Stenosis* is almost invariably a congenital anomaly. It constitutes one of the most important of the congenital cardiac affections. The valve segments are usually united, leaving a small, narrow orifice. In the adult cases occasionally occur. In Case 608 of my post-mortem records there was extreme stenosis in a girl of eighteen, owing to great thickening and adhesion of the segments, and there were also numerous vegetations. The orifice was only two millimetres in diameter. The congenital lesion is commonly associated with patency of the ductus Botalii and imperfection of the ventricular septum. There may also be tricuspid stenosis.

The physical signs are extremely uncertain. There may be a systolic murmur with a thrill heard best to the left of the sternum in the second intercostal space. This murmur may be very like a murmur of aortic stenosis, but is not transmitted into the vessels. Naturally the pulmonary second sound is weak or obliterated, or may be replaced by a diastolic murmur. Usually there is hypertrophy of the right heart.

(*b*) *Pulmonary Insufficiency.*—This rare affection is occasionally due to congenital malformation, particularly fusion of two of the segments. It is sometimes present, as Bramwell has shown, in cases of malignant endocarditis.

The physical signs are those of regurgitation into the right ventricle, but, as a rule, it is impossible to differentiate this from the murmur of aortic insufficiency, though the maximum intensity may be in the pulmonary area. In a recent case, in which two of the valve segments were closely glued to the wall of the pulmonary artery owing to the projection of an aneurism, a diastolic murmur developed under observation, which was transmitted loudly down the sternum. The condition is extremely rare and of little practical significance.

Combined Valvular Lesions.

These are extremely common. The mitral and aortic segments may be affected together; next in frequency comes the combination of mitral and tricuspid lesions; and then of aortic, mitral, and tricuspid. Aortic insufficiency or aortic stenosis is more frequently combined with mitral incompetency than aortic stenosis with mitral stenosis, or mitral stenosis with aortic insufficiency. In children the most common combination is aortic and mitral insufficiency. In adults, mitral insufficiency with thickening of the aortic valves and slight narrowing is perhaps the most common.

The diagnosis rests upon the character of the murmurs and the state of the chambers as regards hypertrophy and dilatation.

Prognosis in Valvular Disease.—The question is entirely one of efficient compensation. So long as this is maintained the patient may suffer no inconvenience, and even with the most serious forms of valve lesion the function of the heart may be little, if at all, disturbed.

Practitioners who are not adepts in auscultation and feel unable to estimate the value of the various heart murmurs should remember that the best judgment of the conditions may be gathered from inspection and palpation. With an apex beat in the normal situation and regular in rhythm the auscultatory phenomena may be practically disregarded.

As Sir Andrew Clark states, a murmur *per se* is of little or no moment in determining the prognosis in any given case. There is a large group of patients who present no other symptoms than a systolic murmur heard over the body of the heart, or over the apex, in whom the left ventricle is not hypertrophied, the heart rhythm is normal, and who may not have had rheumatism. Indeed, the condition is accidentally discovered, often during examination for life insurance. I know cases of this kind which have persisted unchanged for more than fifteen years Among the conditions influencing prognosis are:

(*a*) *Age.*—Children under ten are bad subjects. Compensation is well effected, and they are free from many of the influences which disturb compensation in adults. The coronary arteries also are healthy, and nutrition of the heart-muscle can be readily maintained. Yet, in spite of this, the outlook in cardiac lesions developing in very young children is usually bad. One reason is that the valve lesion itself is apt to be rapidly progressive, and the limit of cardiac reserve force is in such cases early reached. There seems to be proportionately a greater degree of hypertrophy and dilatation. Among other causes of the risks of this period are to be mentioned insufficient food in the poorer classes, the recurrence of rheumatic attacks, and the existence of pericardial adhesions. The outlook in a child who can be carefully supervised and prevented from damaging himself by overexertion is naturally better than in one who is constantly overtasking his muscles. The valvular lesions which develop at, or subsequent to, the period of puberty are more likely to be permanently and efficiently compensated. Sudden death from heart-disease is very rare in children.

(*b*) *Sex.*—Women bear valve lesions, as a rule, better than men, owing partly to the fact that they live quieter lives, partly to the less common involvement of the coronary arteries, and to the greater frequency of mitral lesions. Pregnancy and parturition are disturbing factors, but are, I think, less serious than some writers would have us believe.

(*c*) *Valve affected.*—The relative prognosis of the different valve lesions is very difficult to estimate. Each case must, therefore, be judged on its own merits. Aortic insufficiency is unquestionably the most serious; yet

for years it may be perfectly compensated. Favorable circumstances in any case are the moderate grade of hypertrophy and dilatation, the absence of all symptoms of cardiac distress, and the absence of extensive arterio-sclerosis and of angina. The prognosis rests in reality with the condition of the coronary arteries. Rheumatic lesions of the valves, inducing insufficiency, are less apt to be associated with endarteritis at the root of the aorta; and in such cases the coronary arteries may escape for years. I know a physician, now about thirty-five years of age, who, when sixteen, had his first attack of rheumatism, which involved the aortic segments. He has had two subsequent attacks of rheumatism, but with care has been able to live a comfortable and fairly active life. On the other hand, when the aortic insufficiency is only a part of an extensive arterio-sclerosis at the root of the aorta, the coronary arteries are almost invariably involved, and the outlook in such cases is much more serious. Sudden death is not uncommon, either from acute dilatation during some exertion, or, more frequently, from blocking of one of the branches of the coronary arteries. The liability of this form to be associated with angina pectoris also adds to its severity. Aortic stenosis is a rare lesion, most commonly met with in middle-aged or elderly men, and is, as a rule, well compensated. In many cases it does not appear to limit the duration of life.

In mitral lesions the outlook on the whole is much more favorable than in aortic insufficiency. Mitral insufficiency, when well compensated, carries with it, perhaps, a better prognosis than mitral stenosis; but it must be borne in mind that the cases which last the longest are those in which the valve orifice is more or less narrowed, as well as incompetent. There is, in reality, no valve lesion so rapidly fatal and so poorly compensated as that in which the mitral segments are gradually curled and puckered until they form a narrow strip around a wide mitral ring—a condition specially seen in children. There are many cases of mitral insufficiency in which the defect is thoroughly balanced for thirty or even forty years, without distress or inconvenience. Even with great hypertrophy and the apex beat almost in the mid-axillary line, there may be little or no distress, and the compensation may be most effective. Women may pass safely through repeated pregnancies, though here they are liable to accidents associated with the severe strain. I have had under my care for many years a patient who had her first attack of rheumatism at the age of fifteen, when she already had a well-marked mitral murmur. When she first came under my observation, eighteen years ago, she had signs of hypertrophy of the left ventricle with a loud systolic murmur. She has had no cardiac disturbance whatever. She has lived a very active life, has been unusually vigorous, has borne eleven children, and has passed through three subsequent attacks of rheumatism.

In mitral stenosis the prognosis is usually regarded as less favorable. My own experience has led me, however, to place this lesion almost on a level, particularly in women, with the mitral insufficiency. It is found

very often in persons in perfect health, who have had neither palpitation nor signs of heart-failure, and who have lived laborious lives. The figures given, too, by Broadbent indicate that the date of death in mitral stenosis is comparatively advanced. These patients, too, pass through repeated pregnancies with safety. There are of course those too common accidents, the result of cerebral embolism, which are more liable to occur in this than in other forms.

Hard and fast lines cannot be drawn in the question of prognosis in valvular disease. Every case must be judged separately, and all the circumstances carefully balanced. There is no question which requires greater experience and more mature judgment, and even the most experienced are sometimes at fault.

The following brief summary of the conditions which justify a favorable prognosis embodies the large and varied clinical experience of Sir Andrew Clark: Good general health; just habits of living; no exceptional liability to rheumatic or catarrhal affections; origin of the valvular lesion independently of degeneration; existence of the valvular lesion without change for over three years; sound ventricles, of moderate frequency and general regularity of action; sound arteries, with a normal amount of blood and tension in the smaller vessels; free course of blood through the cervical veins; and, lastly, freedom from pulmonary, hepatic, and renal congestion.

Treatment of Valvular Lesions.—For this purpose the valvular lesion may be divided into the period of progressive development, with establishment and maintenance of hypertrophy, and the period of disturbed compensation.

(*a*) **Stage of Compensation.**—Medicinal treatment at this period is not necessary and is often hurtful. A very common error is to administer cardiac drugs, such as digitalis, on the discovery of a murmur or of hypertrophy. If the lesion has been found accidentally, it may be best not to tell the patient, but rather an intimate friend. Often it is necessary, however, to be perfectly frank in order that the patient may take certain preventive measures. He should lead a quiet, regulated, orderly life, free from excitement and worry. An ordinary wholesome diet should be taken, tobacco should be interdicted, and stimulants not allowed. Exercise should be regulated entirely by the feelings of the patient. So long as no cardiac distress or palpitation follows, moderate exercise will prove very beneficial. The skin should be kept active by a daily bath. Hot baths should be avoided and the Turkish bath should be interdicted. In the case of full-blooded, somewhat corpulent individuals an occasional saline purge should be taken. Patients with valvular lesions should not go into very high altitudes. The act of coition has serious risks, particularly in aortic insufficiency. Knowing that the causes which most surely and powerfully disturb the compensation are overexertion, mental worry, and malnutrition, the physician should give suitable instructions in each

case. As it is always better to have the co-operation of an intelligent patient, he should, as a rule, be told of the condition, but in this matter the physician must be guided by circumstances, and there are cases in which reticence is the wiser policy.

(*b*) **Stage of Broken Compensation.**—The break may be immediate and final, as when sudden death results from acute dilatation or from blocking of a branch of the coronary artery. Among the first indications are shortness of breath on exertion or attacks of nocturnal dyspnœa. These are often associated with impaired nutrition, particularly with anæmia, and a course of iron or change of air may suffice to relieve the symptoms.

Irregularity of the action of the heart cannot always be termed an indication of failing compensation, particularly in instances of mitral disease. It has greater significance in aortic lesions. Serious failure of compensation is indicated by signs of dilatation of the heart, the gallop rhythm, or various forms of arrhythmia, with or without the existence of dropsy. Under these circumstances the following measures are to be carried out:

(1) *Rest.*—Disturbed compensation may be completely restored by rest of the body. Both in Montreal and in Philadelphia it was a favorite demonstration in practical therapeutics to show the influence of complete rest and quiet on the cardiac dilatation. In many cases with œdema of the ankles, moderate dilatation of the heart, and irregularity of the pulse, the rest in bed, a few doses of the compound tincture of cardamoms, and a saline purge suffice, within a week or ten days, to restore the compensation. One patient, in Ward 11 of the Montreal General Hospital, with aortic insufficiency recovered from four successive attacks of failing compensation by these measures alone.

(2) The relief of the embarrassed circulation.

(*a*) *By Venesection.*—In cases of dilatation, from whatever cause, whether in mitral or aortic lesions or distention of the right ventricle in emphysema, when signs of venous engorgement are marked and when there is orthopnœa with cyanosis, the abstraction of from twenty to thirty ounces of blood is indicated. This is the occasion in which timely venesection may save the patient's life. It is a condition in which I have had most satisfactory results from venesection. It is done much better early than late. I have on several occasions regretted its postponement, particularly in instances of acute dilatation and cyanosis in connection with emphysema.*

(*b*) *By Depletion through the Bowels.*—This is particularly valuable when dropsy is present. Of the various purges the salines are to be preferred, and may be given by Matthew Hay's method. Half an hour to an hour before breakfast from half an ounce to an ounce and a half of Epsom salts may be given in a concentrated form. This usually produces

* For illustrative cases from my wards see paper by H. A. Lafleur, Medical News, July, 1891.

^ larger doses are often required.

from three to five liquid evacuations. The compound jalap powder in half-drachm doses, or elaterium, may be employed for the same purpose. Even when the pulse is very feeble these hydragogue cathartics are well borne, and they deplete the portal system rapidly and efficiently.

(*c*) *The Use of Remedies which stimulate the Heart's Action.*—Of these, by far the most important is digitalis, which was introduced into practice by Withering. The indication for its use is dilatation; the contra-indication is a perfectly balanced compensatory hypertrophy, such as we see in all forms of valvular disease. Broken compensation, no matter what the valve lesion may be, is the signal for its use. It acts upon the heart, slowing and at the same time increasing the force of the pulsations. It acts on the peripheral arteries, raising their tension, so that a steady and equable flow of blood is maintained in the capillaries, which, after all, is the prime aim and object of the circulation. The beneficial effects are best seen in cases of mitral disease with small, irregular pulse and cardiac dropsy. Its effects are not less striking in the dilatation of the left ventricle, in the failing compensation of aortic insufficiency or of arterio-sclerosis. On theoretical grounds it has been urged that its use is not so advantageous in aortic insufficiency, since it prolongs the diastole and leads to greater distention. ~~Practically, however,~~ this need not be considered, and, ~~when given with care,~~ digitalis is just as serviceable in this as in any other condition associated with progressive dilatation. It may be given as the tincture or the infusion. In cases of cardiac dropsy, from whatever cause, fifteen minims of the tincture or half an ounce of the infusion may be given every three hours for two days, after which the dose may be reduced. Some prefer the tincture, others the infusion; it is a matter of indifference if the drug is good. The urine of a patient taking digitalis should be carefully estimated each day. As a rule, when its action is beneficial, there is within twenty-four hours an increase in the amount; often the flow is very great. Under its use the dyspnœa is relieved, the dropsy gradually disappears, the pulse becomes firmer, fuller in volume, and sometimes, if it has been very intermittent, regular.

Ill effects sometimes follow digitalis. There is no such thing as a cumulative action of the drug manifested by sudden symptoms. Toxic effects are seen in the production of nausea and vomiting. The pulse becomes irregular and small, and there may be two beats of the heart to one of the pulse, which, as pointed out by Broadbent, is found particularly in cases of mitral stenosis when they are under the influence of this drug. The urine is reduced in amount. These symptoms subside on the withdrawal of the digitalis, and are rarely serious. There are patients who take digitalis uninterruptedly for years, and feel palpitation and distress if the drug is omitted. In mitral disease, even when it does good it does not always steady the pulse. There are many cases in which the irregularity is not affected by the digitalis. When the compensation has been reestablished the drug may be omitted. When there is dyspnœa on exer-

tion and cardiac distress, from five to ten minims three times a day may be advantageously given for prolonged periods, but the effects should be carefully watched. In cardiac dropsy digitalis should be used at the outset with a free hand. Small doses should not be given, but from the first half-ounce doses of the infusion every three hours, or from fifteen to twenty minims of the tincture. There are no substitutes for digitalis.

Of other remedies strophanthus alone is of service. Given in doses of from five to eight minims of the tincture, it acts like digitalis. It certainly will sometimes steady the intermittent heart of mitral valve disease when digitalis fails to do so, but it is not to be compared with this drug when dropsy is present. Convallaria, citrate of caffeine, and *adonis vernalis* are warmly recommended as substitutes for digitalis, but their inferiority is so manifest that their use is rarely indicated.

There are two valuable adjuncts in the treatment of valvular disease—iron and strychnia. When anæmia is a marked feature iron should be given in full doses. In some instances of failing compensation iron is the only medicine needed to restore the balance. Arsenic is occasionally an excellent substitute, and one or other of them should be administered in all instances of heart-trouble when pallor is present. Strychnia is a heart tonic of very great value. It may be given in combination with the digitalis in one or two drop doses of the one per cent solution.

Treatment of Special Symptoms. (*a*) *Dropsy.*—The increased arterial tension and activity of the capillary circulation under the influence of digitalis hastens the interstitial lymph flow and favors resorption of the fluid. The hydragogue cathartics, by rapidly depleting the blood, promote the absorption of the fluid from the lymph spaces and the lymph sacs. These two measures usually suffice to rid the patient of the dropsy. In some cases, however, it cannot be relieved, and then Southey's tubes may be used or the legs punctured. If done with care, after a thorough washing of the parts, and if antiseptic precautions are taken, scarification is a very serviceable measure, and should be resorted to more frequently than it is. Canton-flannel bandages may be applied on the œdematous legs.

(*b*) *Dyspnœa.*—The patients are usually unable to lie down. A comfortable bed-rest should therefore be provided—if possible, one with lateral projections, so that in sleeping the head can be supported as it falls over. The shortness of breath is associated with dilatation, chronic bronchitis, or hydrothorax. The chest should be carefully examined in all these cases, as hydrothorax of one side or of both is a common cause of shortness of breath. There are cases of mitral regurgitation with recurring hydrothorax as the sole dropsical symptom, which is relieved, week by week or month by month, by tapping. For the nocturnal dyspnœa, particularly when combined with restlessness, morphia is invaluable and may be given without hesitation. The value of the calming influence of opium in all conditions of cardiac insufficiency is not enough recognized. There

are instances of cardiac dyspnœa unassociated with dropsy, particularly in mitral-valve disease, in which nitroglycerin is of great service, if given in the one per cent solution in increasing doses. It is especially serviceable in the cases in which the pulse tension is high.

(*e*) *Palpitation and Cardiac Distress.*—In instances of great hypertrophy and in the throbbing which is so distressing in some cases of aortic insufficiency, aconite is of service in doses of from one to three minims every two or three hours. An ice-bag over the heart or Leiter's coil is also of service in allaying the rapid action and the throbbing. For the pains, which are often so marked in aortic lesions, iodide of potassium in ten grain doses, three times a day, or the nitroglycerin may be tried. Small blisters are sometimes advantageous. It must be remembered that an important cause of palpitation and cardiac distress is flatulent distention of the colon, against which suitable measures must be directed.

(*d*) *Gastric Symptoms.*—The cases of cardiac insufficiency which do badly and fail to respond to digitalis are most often those in which nausea and vomiting are prominent features. The liver is often greatly enlarged in these cases; there is more or less stasis in the hepatic vessels, and but little can be expected of drugs until the venous engorgement is relieved. If the vomiting persists it is best to stop the food and give small bits of ice, small quantities of milk and lime water, and effervescing drinks, such as Apollinaris water and champagne. Creosote, hydrocyanic acid, and the oxalate of cerium are sometimes useful; but, as a rule, the condition is obstinate and always serious.

(*e*) *Cough and Hæmoptysis.*—The former is almost a necessary concomitant of cardiac insufficiency, owing to engorgement of the vessels and more or less bronchitis. It is allayed by measures directed rather to the heart than to the lungs. Hæmoptysis in chronic valvular disease is sometimes a salutary symptom. An army surgeon, who was invalided during the late civil war on account of hæmoptysis, supposed to be due to tuberculosis, has since that time had, in association with mitral insufficiency and enlarged heart, many attacks of hæmoptysis. He assures me that his condition is invariably better after the attack. It is rarely fatal, except in some cases of acute dilatation, and seldom calls for special treatment.

(*f*) *Sleeplessness.*—One of the most distressing features of valvular lesions, even in the stage of compensation, is disturbed sleep. Patients may wake suddenly with throbbing of the heart, often in an attack of nightmare. Subsequently, when the compensation has failed, it is also a worrying symptom. The sleep is broken, restless, and frequently disturbed by frightful dreams. Sometimes a dose of the spirits of chloroform or of ether, with half a drachm of spirits of camphor, given in a little hot whisky, will give a quiet night. The compound spirits of ether, Hoffman's anodyne, though very unpleasant to take, is frequently a great boon in the intermediate period when compensation has partially failed

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and the patients suffer from restless and sleepless nights. Paraldehyde and amylene hydrate are sometimes serviceable. Urethan, sulphonal, and chloralamide are rarely efficacious, and it is best, after a few trials, particularly if the paraldehyde does not answer, to give morphia. It may be given in combination with atropine.

(*g*) *Renal Symptoms.*—With ruptured compensation and lowering of the tension in the aorta, the urinary secretion is greatly diminished, and the amount may sink to five or six ounces in the day. Digitalis and strophanthus, when efficient, usually increase the flow. A brisk purge may be followed by augmented secretion. The combination in pill form of digitalis, squill, and the black oxide of mercury, will sometimes prove effective when the infusion or tincture of digitalis alone has failed. Calomel acts well in some cases, given in grs. iij every six hours for three or four days.

The *diet* in chronic valve diseases is often very difficult to regulate. With the dilatation and venous engorgement come nausea and often a great distaste for food. The amount of liquid should be restricted, and milk, beef-juice, or egg albumen given every three hours. When the serious symptoms have passed, eggs, scraped meat, fish, and fowl may be allowed. Starchy foods, and all articles likely to cause flatulency, should be forbidden. Stimulants are usually necessary, either whisky or brandy.

III. HYPERTROPHY AND DILATATION.

Hypertrophy is an enlargement of the heart due to an increased thickness, total or partial, in the muscular walls. Dilatation is an increase in size of one or more of the chambers with or without thickening of the walls. The conditions usually coexist, and could be more correctly described together under the term enlargement of the heart. Simple hypertrophy, in which the cavities remain of a normal size and the walls are increased, occurs, but simple dilatation, in which the cavities are increased and the walls remain of a normal diameter, probably does not, as it is always associated with thinning or with thickening of the coats. Commonly we have the forms of simple hypertrophy, hypertrophy with dilatation, and dilatation with thinning of the coats.

Hypertrophy of the Heart.

There are two forms—the simple hypertrophy, in which the cavity or cavities are of normal size; and hypertrophy with dilatation (eccentric hypertrophy), in which the cavities are enlarged and the walls increased in thickness. The condition formerly spoken of as concentric hypertrophy, in which there is diminution in the size of the cavity with thickening of the walls, is, as a rule, a post-mortem change.

The enlargement may affect the entire organ, one side, or only one chamber. Naturally, as the left ventricle does the chief work in forcing the blood through the systemic arteries, the change is most frequently found in it.

Etiology.—Hypertrophy of the heart follows the law governing muscles, that within certain limits, if the nutrition is kept up, increased work is followed by increased size—i. e., hypertrophy. Hypertrophy of the left ventricle alone, or with general enlargement of the heart, is brought about by—

Conditions affecting the heart itself: (1) Disease of the aortic valve; (2) mitral insufficiency; (3) general pericardial adhesions; (4) sclerotic myocarditis; (5) disturbed innervation, with overaction, as in exophthalmic goitre, in long-continued nervous palpitation, and as a result of the action of certain articles, such as tea, alcohol, and tobacco. In all of these conditions the work of the heart is increased. In the case of the valve lesions the increase is due to the increased intraventricular pressure; in the case of the adherent pericardium and myocarditis, to direct interference with the symmetrical and orderly contraction of the chambers.

Conditions acting upon the blood-vessels: (1) General arterio-sclerosis, with or without renal disease; (2) all states of increased arterial tension induced by the contraction of the smaller arteries under the influence of certain toxic substances, which act, as Bright suggested, by affecting "the minute capillary circulation, render greater action necessary to send the blood through the distant subdivisions of the vascular system"; (3) prolonged muscular exertion, which enormously increases the blood-pressure in the arteries; (4) narrowing of the aorta, as in the congenital stenosis.

Hypertrophy of the right ventricle is met with under the following conditions—

(1) Lesions of the mitral valve, either incompetence or stenosis, which act by increasing the resistance in the pulmonary vessels. (2) Pulmonary lesions, obliteration of any number of blood-vessels within the lungs, such as occurs in emphysema or cirrhosis, is followed by hypertrophy of the right ventricle. (3) Valvular lesions on the right side occasionally cause hypertrophy in the adult, not infrequently in the fœtus. (4) Chronic valvular disease of the left heart and pericardial adhesions are sooner or later associated with hypertrophy of the right ventricle.

In the auricles simple hypertrophy is never seen; it is always dilatation with hypertrophy. In the left auricle the condition develops in lesions at the mitral orifice, particularly stenosis. The right auricle hypertrophies when there is greatly increased blood-pressure in the lesser circulation, whether due to mitral stenosis or pulmonary lesions. Narrowing of the tricuspid orifice is a less frequent cause.

Morbid Anatomy.—The heart of an average-sized man weighs about nine ounces (280 grammes); that of a woman, about eight ounces (250 grammes). In cases of general hypertrophy the heart may weigh

from sixteen to twenty ounces. Weights above twenty-five ounces are rare. So far as I know, the heaviest heart on record is one described by Beverly Robinson, weighing fifty-three ounces. Dulles has reported one weighing forty-eight ounces. The measurement of the thickness of the walls is, next to weighing, the best means of determining the hypertrophy. In extreme dilatation the walls, though actually thickened, may look thin. When *rigor mortis* is present, the cavity may be small and the walls may appear greatly thickened. The measurements should not be made until the heart has been soaked in water and thoroughly relaxed. In the left ventricle a thickness of ten lines, or from twenty to twenty-five millimetres, indicates hypertrophy. The right ventricle is thinner than the left, and has an average diameter of from four to seven millimetres. In hypertrophy it may measure from thirteen to twenty millimetres. The left auricle has a normal thickness of about three millimetres, which may be doubled in hypertrophy. The wall of the right auricle is thinner than that of the left, rarely exceeding two millimetres in diameter. The appendices of the auricles often present marked increase in thickness and the musculi pectinati are greatly developed.

The shape of the heart is altered in hypertrophy; with great enlargement of the ventricles, the apex is broadened, and the conical shape is lost. In the enormous enlargement of aortic insufficiency this rotundity of the apex is very marked. When the right ventricle is chiefly affected it occupies the largest share of the apex. In mitral stenosis the contrast is very striking between the large, broad right ventricle, reaching to the apex, and the small left chamber.

The hypertrophied muscle has a deep red color, is firm, and is cut with increasing resistance. The right ventricle, as Rokitansky noted, may have a peculiar hard, leathery consistence. In simple hypertrophy of the left ventricle the papillary muscles and the columnæ carneæ may be enlarged, but the former are often much flattened in dilated hypertrophy. The muscular trabeculæ are more developed, as a rule, in the right ventricle than in the left.

The increase in size of the heart is probably due to a definite numerical increase, resulting from development of new fibres.

Symptoms.—Hypertrophy is a conservative process, secondary to some valvular or arterial lesion, and is not necessarily accompanied by symptoms. So admirable is the adjusting power of the heart that, for example, an advancing stenosis of aortic or mitral orifice may for years be perfectly equalized by a progressive hypertrophy, and the subject of the affection be happily unconscious of the existence of heart-trouble. Hypertrophy is in almost all cases an unmixed good; the symptoms which arise are usually to be attributed to its failure, or, as we say, to disturbance of compensation.

Among the most common symptoms are unpleasant feelings about the heart—a sense of fulness and discomfort, rarely amounting to pain. This

may be very noticeable when the patient is recumbent on the left side. Actual pain is rare, except in the irritable heart from tobacco or in neurasthenics. Palpitation may not occur, nor do patients always have sensations from the violent shocks of a greatly hypertrophied organ. There are instances in which very uneasy feelings arise from a moderately exaggerated pulsation. The general condition has much to do with this. In health we are not conscious of the heart's pulsations, but one of the first indications of exhaustion from excesses or overstudy is the consciousness of the heart's action, not necessarily with palpitation. Headaches, flushings of the face, noises in the ears, and flashes of light may be present.

Certain untoward effects of long-continued hypertrophy of the left ventricle must be mentioned, chief among which is the production of arterio-sclerosis. Particularly is this the case when the hypertrophy results from increased peripheral resistance. The heightened blood-pressure (expressed by the word strain) in the arteries gradually induces an endarteritis and a stiff, inelastic state of those vessels most exposed to it—viz., the aorta and its primary divisions. In overcoming the peripheral obstruction the hypertrophy "ruins the arteries as a sequential result" (Fothergill). Prolonged muscular exertion also acts injuriously in this way.

Another danger is rupture of the blood-vessels, particularly those of the brain. In general arterial degeneration associated with contracted kidneys and hypertrophied left heart apoplexy is common. Indeed, in the majority of cases of cerebral hæmorrhage there is sclerosis of the smaller vessels, often with the development of miliary aneurisms, and the rupture may be caused by the forcible action of the heart.

Physical Signs.—*Inspection* may show bulging of the præcordia, producing in children marked asymmetry of the chest. It may occur without pericardial adhesions, which Schroetter thinks are invariably associated with this condition. The intercostal spaces are widened, and the area of visible impulse is much increased. On *palpation* the impulse is forcible and heaving, and with each systole the hand or the ear applied over the heart may be visibly raised. A slow, heaving impulse is one of the best signs of simple hypertrophy. With large dilated hypertrophy the forcible impulse is often more sudden and abrupt. A second, weaker impulse can sometimes be felt, due perhaps to a rebound from the aortic valves (Gowers). The beat may be felt in the sixth, seventh, or eighth interspace from one to three inches outside the nipple. This downward dislocation of the apex is an important sign in hypertrophy of the left ventricle. In moderate grades, such as are seen in chronic Bright's disease, the impulse may be in the sixth interspace in the nipple line, or a little outside of it.

Percussion reveals increased dulness, which in the parasternal line may begin at the third rib or in the second interspace, and transversely may extend from half an inch to two inches beyond the nipple line and an equal distance beyond the middle line of the sternum. The dull area

is more ovoid than in health. When carefully delimited the colossal hypertrophy of aortic valve disease may give an area of dulness from seven to eight inches in transverse extent. In moderate grades a transverse dulness of four inches is not uncommon.

On *auscultation* the sounds, when the valves are healthy, may present no special changes, but the first sound is often prolonged and dull. When there is dilatation as well, it may be very clear and sharp. Reduplication is common in the hypertrophy of renal disease. A peculiar clink —the *tintement métallique* of Bouillaud—may be heard just to the right of the apex beat. The second sound is clear and loud, sometimes ringing in character or reduplicated. With valvular lesions, the sounds, of course, are much altered, and are replaced or accompanied by murmurs.

In simple hypertrophy not dependent on valvular lesions, the pulse is usually regular, full, strong, and of high tension. It may be increased in rapidity, but is often normal. In eccentric hypertrophy the pulse is full, but softer, and usually more rapid. One of the earliest signs of failure and dilatation is irregularity and intermittence of the pulse.

Hypertrophy of the *right ventricle* in the adult very rarely follows valvular disease on the right side, but results from increased resistance in the pulmonary circulation, as in cirrhosis of the lung and emphysema, or in stenosis of the mitral orifice. With perfect compensation, which fully maintains the equilibrium of the circulation, there are no symptoms. Extra exertion, as the ascent of stairs or running, may cause shortness of breath, but in many ways hypertrophy of the right ventricle is the most enduring and salutary form in the whole cycle of cardiac affections. For long periods of years the effects of mitral stenosis may be counterbalanced, and only sudden death by accident or an acute disease reveal the existence of an unsuspected lesion. In the hypertrophy secondary to emphysema or cirrhosis of the lungs, there may be sensations of distress in the cardiac region, with cough and shortness of breath; but as long as the dilatation is moderate the symptoms are not marked. With great dilatation and tricuspid leakage come venous engorgement, œdema, and pulmonary troubles. The increased pressure in the lesser circulation leads to sclerosis of the pulmonary arteries and the constant engorgement of the capillaries leads ultimately to a deposition of pigment and increase in the fibrous elements in the lung—the brown induration. Extreme pulmonary congestion and apoplexy are more often associated with dilatation. Hæmoptysis may result from rupture of vessels during sudden exertion.

Physical Signs.—Bulging of the lower part of the sternum and left cartilages occurs. The apex beat is forced to the left, but is not so often displaced downward. The most marked impulse may be in the angle between the ensiform cartilage and the seventh rib or beneath the cartilages of the sixth and seventh ribs. The pulsation is rather diffuse, not punctuate, particularly if there is much dilatation. In thin-walled chests there may be pulsation in the third and fourth right interspaces.

and to a marked grade in many congenital affections of the heart.

The cardiac dulness is increased transversely and toward the right; it may extend an inch or more beyond the border of the sternum. On auscultation the first sound at the lower part of the sternum is louder and fuller than normal, but the differences are not very marked unless there is much dilatation, when the sound is clearer and sharper. Accentuation and reduplication of the second sound are heard in the pulmonary artery on account of the increased tension. The pulse at the wrist is usually small. Pulsation occurs in the jugulars when there is tricuspid incompetence.

Hypertrophy of the *auricles* always occurs with dilatation. It is most common in the left chamber, which hypertrophies in mitral stenosis and incompetency and naturally assists in restoring the balance of the circulation. There are no distinctive physical signs, and we usually can infer its presence only by the existence of mitral stenosis and a presystolic murmur. Increased dulness may be determined to the left of the sternum, and there may be a presystolic wave in the second left interspace.

Hypertrophy and dilatation of the right auricle are met with (associated with a similar condition in the right ventricle and incompetency of the tricuspid) in emphysema, cirrhosis of the lung, chronic bronchitis, and mitral disease. In comparison with the left auricle the greater development and hypertrophy of the appendix and its musculi pectinati is very striking. The latter may be distributed over the anterior wall of the sinus to a greater extent than in health. There are increased dulness in the third and fourth interspaces, pulsation sometimes presystolic in rhythm, signs of venous engorgement, jugular pulsation, and other evidences of dilatation of the right heart.

Diagnosis.—Among conditions to be distinguished are:

(1) Neurotic palpitation, from whatever cause, even when very forcible, has not the heaving impulse of genuine hypertrophy. Enlargement of the organ may, however, follow prolonged overaction, as in the smoker's heart, the irritable heart of neurasthenics, and in exophthalmic goitre, but it is usually slight.

(2) The increased area of dulness may be due to a variety of causes, some of which may closely simulate hypertrophy, such as pericardial effusion, aneurism, mediastinal growths, or displacement of the heart from pressure, or the existence of malformation of the chest. With the exercise of ordinary care, however, the diagnosis can usually be made. There are two opposite conditions which frequently give trouble. With the left lung contracted from pleurisy, phthisis, or cirrhosis, a large surface of the heart is exposed; the pulsation may be extensive and forcible, and may at first sight resemble hypertrophy. In this condition there is dislocation upward and to the left. The existence of pulmonary or pleuritic disease and the fixation of the lung on deep inspiration will suffice to prevent mistakes. A less extensive exposure of the heart may occur without any disease in very narrow-chested persons with ill-developed lungs; here, though the area of dulness may be much increased, the normal position

of the apex, the absence of forcible, heaving impulse, and of any obvious cause of hypertrophy will afford satisfactory criteria for a diagnosis. The reverse condition exists in some cases in which emphysema masks moderate cardiac hypertrophy. The area of dulness may be normal, or even diminished, and the pulse and character of the sounds will help in the diagnosis; but it is sometimes a difficult matter.

Prognosis.—The course of any case of cardiac hypertrophy may be divided into three stages:

(*a*) The period of development, which varies with the nature of the primary lesion. For example, in rupture of an aortic valve, during a sudden exertion, it may require months before the hypertrophy becomes fully developed; or, indeed, it may never do so, and death may follow from an uncompensated dilatation. On the other hand, in sclerotic affections of the valves, with stenosis or incompetency, the hypertrophy develops step by step with the lesion, and may continue to counterbalance the progressive and increasing impairment of the valve.

(*b*) The period of full compensation—the latent stage—during which the heart's vigor meets the requirements of the circulation. This period may last an indefinite time, and a patient may never be made aware by any symptoms that he has a valvular lesion.

(*c*) The period of broken compensation, which may come on suddenly during very severe exertion. Death may result from acute dilatation; but more commonly it takes place slowly and results from degeneration and weakening of the heart-muscle.

The breaking or rupture of cardiac compensation may be induced by many causes, among which the most important are: (1) Failure of the general nutrition. In many instances of heart-disease, exposure, poor food, and alcohol combine to bring about disturbance of a well-balanced heart lesion. Acute illnesses, particularly the fevers, may induce general debility and with it weakening of the heart-muscle. (2) Disturbance of the local nutrition of the heart, owing to gradual sclerosis of the coronary arteries, is a common cause. (3) Very severe muscular exertion, which may disturb a compensation, perfect for years, and induce death in a few days (Traube). (4) Mental emotions. Severe grief or fright may bring on failure of compensation.

The prognosis is largely, as already stated, a matter of maintained compensation. Once established, the hypertrophy rarely, if ever, disappears, inasmuch as the cause usually persists. Occasionally, perhaps, the hypertrophy associated with neurotic palpitation from tobacco, or other causes, or the hypertrophy following muscular over-exertion, may disappear.

Dilatation of the Heart.

Two varieties are recognized, dilatation with thickening and dilatation with thinning. The former is the most common, and corresponds to the dilated or eccentric hypertrophy.

Etiology.—Two important causes combine to produce dilatation—increased pressure within the cavities and impaired resistance, due to weakening of the muscular wall—which may act singly, but are often combined. A weakened wall may yield to a normal distending force, or a normal wall may yield under a heightened blood-pressure.

(1) Heightened endocardiac pressure results either from an increased quantity of blood to be moved or an obstacle to be overcome, and is the most frequent cause. It does not necessarily bring about dilatation; simple hypertrophy may follow, as in the early period of aortic stenosis, and in the hypertrophy of the left ventricle in Bright's disease.

A majority of the important causes of increased endocardiac pressure have already been discussed under hypertrophy. One or two may be considered more in detail.

The size of the cardiac chambers varies in health. With slow action of the heart the dilatation is complete and fuller than it is with rapid action. Physiologically, the limits of dilatation are reached when the chamber does not empty itself during the systole. This may occur as an acute, transient condition in severe exertion—during, for example, the ascent of a mountain. There may be great dilatation of the right heart, as shown by the increased epigastric pulsation, and even increase in the cardiac dulness. The safety-valve action of the tricuspid valves may here come into play, relieving the lungs by permitting regurgitation into the auricle. With rest the condition is removed, but if it has been extreme, the heart may suffer a strain from which it may recover slowly, or, indeed, the individual may never be able again to undertake severe exertion. In the process of training, the getting wind, as it is called, is largely a gradual increase in the capability of the heart, particularly of the right chambers. A degree of exertion can be safely maintained in full training which would be quite impossible under other circumstances, because by a gradual process of what we may call physical education the heart has strengthened its reserve force—widened enormously its limits of physiological work. Endurance in prolonged contests is measured by the capabilities of the heart, and its essence consists in being able to meet the continuous tendency to overstep the limits of dilatation.

We have no positive knowledge of the nature of the changes in the heart which occur in this process, but it must be in the direction of increased muscular and nervous energy. The large heart of athletes may be due to the prolonged use of their muscles, but no man becomes a great runner or oarsman who has not naturally a capable if not a large heart. Master McGrath, the celebrated greyhound, and Eclipse, the

race-horse, both famous for endurance rather than speed, had very large hearts.

Excessive dilatation during severe muscular effort results in heart-strain. A man, perhaps in poor condition, calls upon his heart for extra work during the ascent of a high mountain, and is at once seized with pain about the heart and a sense of distress in the epigastrium. He breathes rapidly for some time, is "puffed," as we say, but the symptoms pass off after a night's quiet. An attempt to repeat the exercise is followed by another attack, or, indeed, an attack of cardiac dyspnœa may come on while he is at rest. For months such a man may be unfitted for severe exertion, or he may be permanently incapacitated. In some way he has overstrained his heart and become "broken-winded." Exactly what has taken place in these hearts we cannot say, but their reserve force is lost, and with it the power of meeting the demands exacted in maintaining the circulation during severe exertion. The "heart-shock" of Latham includes cases of this nature—sudden cardiac breakdown during exertion, not due to rupture of a valve. It seems probable that sudden death in men during long-continued efforts, as in a race, is sometimes due to over-distention and paralysis of the heart.

Examples of dilatation occur in all forms of valve lesions. In aortic incompetency blood enters the left ventricle during diastole from the unguarded aorta and from the left auricle, and the quantity of blood at the termination of diastole subjects the walls to an extreme degree of pressure, under which they inevitably yield. In time they augment in thickness, and present the typical eccentric hypertrophy of this condition.

In mitral insufficiency blood which should have been driven into the aorta is forced into and dilates the auricle from which it came, and then in the diastole of the ventricle a large amount is returned from the auricle, and with increased force. In mitral stenosis the left auricle is the seat of greatly increased tension during diastole, and dilates as well as hypertrophies; the distention, too, may be enormous. Dilatation of the right ventricle is produced by a number of conditions, which were considered under hypertrophy. All circumstances, such as mitral stenosis, emphysema, etc., which permanently increase the tension of the blood in the pulmonary vessels, will cause its dilatation.

(2) Impaired nutrition of the heart-walls may lead to a diminution of the resisting power so that dilatation readily occurs.

The loss of tone due to parenchymatous degeneration or myocarditis in fevers may lead to a fatal condition of acute dilatation. It is a recognized cause of death in scarlatinal dropsy (Goodhart), and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. The changes in the heart-muscle which accompany acute endocarditis or pericarditis may lead to dilatation, especially in the latter disease. In anæmia, leukæmia, and chlorosis the dilatation may be considerable. In sclerosis of the walls, the yielding is always where this process is most advanced, as at the left apex.

Under any of these circumstances the walls may yield with normal blood-pressure.

Pericardial adhesions are a cause of dilatation, and we generally find in cases with extensive and firm union considerable hypertrophy and dilatation. There is usually here some impairment as well of the superficial layers of muscle.

Morbid Anatomy.—The condition usually exists with hypertrophy in two or more chambers. It is more common on the right than on the left side. The most extreme dilatation is in cases of aortic incompetency, in which all the cavities may be enormously distended. In mitral stenosis the left auricle is often trebled in capacity, and the right chambers also are very capacious. The auricles may contain from eighteen to twenty ounces of blood. In chronic lesions of the lungs the right chambers are chiefly involved. In great distention of one ventricle the septum may bulge toward the other side. The auriculo-ventricular rings are often dilated, and there may be an increase in the circumference of an inch and a half or even two inches. Thus, the tricuspid orifice, the circumference of which is about four and a half inches, may freely admit a graduated heart-cone of over six inches; and the mitral orifice, which normally is about three and a half inches, may admit the cone to five and a half inches or even more. Great dilatation is always accompanied with relative incompetency of the valves, so that free regurgitation into the auricles is permitted. The orifices of the venæ cavæ and of the pulmonary veins may be greatly dilated.

The endocardium is often opaque, particularly that of the auricles. The muscle substance varies according to the presence or absence of degenerations. The microscope may show marked fatty or parenchymatous change, but in some instances no special alteration may be noticeable. There is much truth in Niemeyer's assertion "that it is not possible by means of the microscope to recognize all the alterations of the muscular fibrillæ which diminish the functional power of the heart." Of the changes in the ganglia of the heart we know very little. As centres of control they probably have more to do with cardiac atony and breakdown than we generally admit. Degeneration of them has been noted by Putjakin, Ott, and others.

Symptoms and Physical Signs.—Dilatation causes weakness of the cardiac walls, diminishes the vigor of their contractions, and is therefore the reverse of hypertrophy. So long as compensation is maintained the enlargement of a cavity may be considerable. The limit is reached when the hypertrophied walls in the systole can no longer expel all the contents, part of which remain, so that at each diastole the chamber is abnormally full. Thus, in aortic incompetency blood enters the left ventricle from the aorta as well as the auricle; dilatation ensues, and also hypertrophy as a direct effect of the increased pressure and increased amount of blood to be moved. But if from any cause the hypertrophy

weakens and the ventricle during systole fails to empty itself completely, a still larger amount is in it at the end of each diastole, and the dilatation becomes greater. The amount remaining after systole prevents the blood from entering freely from the auricle. Incompetency of the auriculo-ventricular valves follows, with dilatation of the auricle and impeded blood-flow in the pulmonary veins. Dilatation and hypertrophy of the right heart may compensate for a time, but when this fails the venous system becomes engorged and dropsy may result. The consideration of the symptoms of chronic valvular lesions is largely that of dilatation and its effects. Acute dilatation, such as we see in fevers or in sudden failure of a hypertrophied heart, is accompanied by three chief symptoms—weak, usually rapid, impulse, dyspnœa, and signs of obstructed venous circulation. Cardiac pain may be present, but is often absent.

The *physical signs* of dilatation are those of a weak and enlarged organ. The impulse is diffuse, often undulatory, and is felt over a wide area, and an apex beat or a point of maximum intensity may not exist. When it does exist, it may be visible and yet cannot be felt—a valuable observation made by Walshe. An extensive area of impulse with a quick, weak maximum apex beat may be present. When the right heart is chiefly dilated the left may be pushed over so as to occupy a much less extensive area in front of the heart, and the true apex beat cannot be felt; but the chief impulse is just below, or to the right of, the xiphoid cartilage, and there is a wavy pulsation in the fourth, fifth, and sixth interspaces to the left of the sternum. In extreme dilatation of the right auricle a pulsation may sometimes be seen in the third right interspace close to the sternum, and with free tricuspid regurgitation this may be systolic in character. Whether the pulsation frequently seen in the second left interspace is ever due to a dilated left auricle has not been determined. I have sometimes thought it was presystolic in rhythm, though it may be distinctly systolic. Post mortem, it is rare in the most extreme distention to see the auricular appendix so far forward as to warrant the belief that it could beat against the second interspace. The area of dulness is increased, but an emphysematous lung or the fully distended organ in a state of brown induration may cover over the heart and greatly limit the extent. The directions of increase were considered in connection with hypertrophy.

The first sound is shorter, sharper, more valvular in character, and more like the second. As the dilatation becomes excessive it gets weaker. Reduplication is not common, but occasionally differences may be heard in the first sound over the right and left hearts. The sounds are frequently obscured by murmurs, which are produced by incompetency of the valves due to the great dilatation, or are associated with the chronic valve disease on which the condition depends. The aortic second sound is replaced by a murmur in aortic regurgitation. The pulmonary sound is accentuated in mitral regurgitation and pulmonary congestion, but

with extreme dilatation it may be much weakened. The heart's action is irregular and intermittent, and the pulse is small, weak, and quick.

On auscultation both the sounds may be free from murmur. Often there is the condition known as embryocardia or fœtal heart-rhythm, in which the first and second sounds are very alike, and the long pause is shortened. In other instances there is the typical and characteristic gallop rhythm, rarely found apart from conditions of dilatation. With the various valvular lesions the corresponding murmurs may be heard. Murmurs, however, which have been present may disappear, as in the case of mitral stenosis. In other instances a loud systolic murmur may be heard at the apex, and when the case first comes under observation it may be impossible to say whether this is due to organic mitral lesion. The murmur may be confined to the apex region, or propagated well to the back. It is extremely common in the dilatation which follows the hypertrophy of the left ventricle in arterio-sclerosis. Under treatment, with the gradual disappearance of the dilatation, a murmur of this kind, even though most intense, may completely disappear, showing that it has been due to a relative insufficiency, not to a valvular lesion. All varieties of arrhythmia may occur in dilatation of the heart. The pulse, as a rule, is small, weak, quick, and often irregular.

Dilatation and Hypertrophy due to Overexertion and Alcohol.—There is a group of cases of dilatation and hypertrophy dependent upon prolonged overexertion, which rarely comes under observation until compensation has failed, and which then may be very difficult to distinguish from the similar conditions produced by valvular disease. The patients are able-bodied men at the middle period of life, and complain first of palpitation or irregularity of the action of the heart, shortness of breath, and subsequently the usual symptoms of cardiac insufficiency develop. On inquiring into the history of these patients none of the usual etiological factors causing valve disease are present, but they have always been engaged in laborious occupations and have usually been in the habit of taking stimulants freely. This is the affection which has been specially studied by McLean, Clifford Albutt, Seitz, and others, and in its earlier condition by Da Costa, in what he termed the irritable heart. It is met with very frequently in soldiers. These cases may return to hospital three or four times with cardiac insufficiency, sometimes with slight anasarca, hæmoptysis, and signs of pulmonary engorgement. The condition is by no means infrequent. Bollinger has called attention to the common occurrence of dilatation and hypertrophy in beer-drinkers, particularly in the workers in the German breweries, who drink twenty or more litres in the day. Strümpell, at his Erlangen clinic, told me that this condition was very common in the draymen and workers in the breweries of that town, very few of whom pass the forty-fifth year without indications of hypertrophy and dilatation of the heart. On post-mortem examination the valves may be quite healthy, the aorta smooth, and no extensive arterio-

sclerosis or renal disease. The heart weighs from eighteen to twenty-five ounces; the chambers are dilated. The condition has been met with also in animals, and Houghton states that the heart of the celebrated greyhound Master McGrath weighed 9·57 ounces, just threefold in excess of the normal proportion of heart-weight to body-weight.

Idiopathic Dilatation.—And, lastly, there are other cases in which dilatation of the heart occurs without discoverable cause. In some instances there has been a history of sudden exercise or of mental emotion, but in other cases the condition seems to have come on spontaneously. In some the condition is acute and the patient has dyspnœa, slight cyanosis, cough, and great cardiac distress. Death may occur in a few days, or dropsy may supervene and the case may become chronic. Delafield has reported an interesting series of cases of this group.

Treatment.—The treatment of hypertrophy and dilatation has already been considered under the section on valvular lesions. I would only here emphasize the fact that with signs of dilatation, as indicated by gallop rhythm, urgent dyspnœa, and slight lividity, venesection is in many cases the only means by which the life of the patient may be saved, and from twenty-five to thirty ounces of blood should be abstracted without delay. Subsequently stimulants, such as ammonia and digitalis, may be administered, but they are accessories only to the bleeding in the critical condition of acute dilatation, which is so frequently met with in cardiac lesions.

IV. AFFECTIONS OF THE MYOCARDIUM.

1. **Lesions due to Disease of the Coronary Arteries.**—A knowledge of the changes produced in the myocardium by disease of the coronary vessels gives a key to the understanding of many problems in cardiac pathology. The terminal branches of the coronary vessels are end arteries. The blocking of one of these vessels by a thrombus or an embolus leads to a condition which is known as—

(*a*) *Anæmic necrosis*, or white infarct. This is most commonly seen in the left ventricle and in the septum, in the territory of distribution of the anterior coronary artery. The affected area has a yellowish-white color, sometimes a turbid, parboiled aspect, at others a grayish-red tint. It may be somewhat wedge-shaped, more often it is irregular in contour and projects above the surface. Microscopically the changes are very characteristic. The nuclei disappear from the muscle fibres, the condition of fragmentation is present, and the fibres present a homogeneous, hyaline appearance. In some instances there is complete transformation, and even to the naked eye a firm white patch of hyaline degeneration may appear in the centre of the area. Sudden death not infrequently follows the blocking of one of the branches of the coronary

artery and the production of this anæmic necrosis. *In medico-legal cases it is a point of primary importance to remember that this is one of the common causes of sudden death.* This condition should be carefully sought for, inasmuch as it may be the sole lesion, except a general, sometimes slight arterio-sclerosis. Rupture of the heart may be associated with anæmic necrosis.

(*b*) The second important effect of coronary-artery disease upon the myocardium is seen in the production of *fibrous myocarditis.* This may result from the gradual transformation of areas of anæmic necrosis. More commonly it is caused by the narrowing of a coronary branch in a process of obliterative endarteritis. The sclerosis is most frequently seen at the apex of the left ventricle and in the septum, but it may occur in any portion. In the septum often there are streaks of fibroid degeneration which do not reach the endocardium, and it may be necessary to divide the muscle in order to see them. Hypertrophy of the heart is commonly associated with this degeneration. It is the invariable precursor of aneurism of the heart.

Complete obliteration of one coronary artery, if produced suddenly, is usually fatal. When induced slowly, either by arterio-sclerosis at the orifice of the artery at the root of the aorta or by an obliterating endarteritis in the course of the vessel, the circulation may be carried on through the other vessel. Sudden death is not uncommon, owing to thrombosis of a vessel which has become narrowed by sclerosis. In the most extreme grade one coronary artery may be entirely blocked, with the production of extensive fibroid disease, and a main branch of the other also may be occluded. A large, powerfully built imbecile, aged thirty-five, at the Elwyn Institution, Pennsylvania, who had for years enjoyed doing the heavy work about the place, died suddenly, without any preliminary symptoms. The heart, which is in my collection, weighed over twenty ounces; the anterior coronary artery was practically occluded by obliterating endarteritis, and of the posterior artery one main branch was occluded.

(*c*) *Septic Infarcts.*—In pyæmia the smaller branches of the coronary arteries may be blocked with septic emboli and cause infarcts in the myocardium in the form of ~~miliary~~ abscesses, varying in size from a pea to a pin's head. These may not cause any disturbance, but when large they may perforate into the ventricle or into the pericardium, forming what has been called acute ulcer of the heart.

2. **Acute Interstitial Myocarditis.**—In the fevers and in pericarditis the intermuscular connective tissue is swollen and infiltrated with round cells and nuclei, the vessels are dilated, there are minute extravasations, and the muscle fibres may be granular or fatty, with indistinct striæ and nuclei. These instances have been met with in typhoid fever, smallpox, and diphtheria. The muscle substance is pale, soft, and easily torn, and the condition has been described either as inflammatory or degenerative.

3. **Parenchymatous Degeneration.**—This is usually met with in fevers, or in connection with endocarditis or pericarditis. It is characterized by a pale, turbid state of the cardiac muscle, which is general, not localized. Turbidity and softness are the special features. It is the softened heart of Laennec and Louis. Stokes speaks of an instance in which "so great was the softening of the organ that when the heart was grasped by the great vessels and held with the apex pointing upward, it fell down over the hand, covering it like a cap of a large mushroom."

Histologically, there is a degeneration of the muscle fibres, which are infiltrated to a various extent with granules which resist the action of ether. Sometimes this granular change in the fibres is extreme, and no trace of the striæ can be detected. It is probably the effect of a toxic agent, and is seen in its most exquisite form in the lumbar muscles in cases of toxic hæmoglobinuria in the horse. It is met with in cases of typhoid, typhus, small-pox, and other infectious diseases, particularly when the course is protracted. There is no definite relation between it and the high temperature.

A form of myocarditis has been described, characterized by fragmentation of the fibres owing to softening of the cement substance. According to von Recklinghausen this is a post-mortem change.

4. **Fatty Heart.**—Under this term are embraced fatty degeneration and fatty overgrowth.

(*a*) *Fatty degeneration* is a very common condition, and mild grades are met with in many diseases. It is found in the failing nutrition of old age, of wasting diseases, and of cachectic states; in prolonged infectious fevers, in which it may follow or accompany the parenchymatous change; associated with acute and chronic anæmias. Certain poisons, such as phosphorus, produce an intense fatty degeneration. Local causes: Pericarditis is usually associated with fatty or parenchymatous changes in the superficial layers of the myocardium. Disease of the coronary arteries is a common and important cause. Lastly, in the hypertrophied ventricular wall in chronic heart-disease fatty change is by no means infrequent. This degeneration may be limited to the heart or it may be more or less general in the solid viscera. The diaphragm may also be involved, even when the other muscles show no special changes. There appears to be a special proneness to fatty degeneration in the heart-muscle, which may perhaps be connected with its incessant activity. So great is its need of an abundant oxygen supply that it feels at once any deficiency, and is in consequence the first muscle to show nutritional changes.

Anatomically the condition may be local or general. The left ventricle is most frequently affected. If the process is advanced and general the heart looks large and is flabby and relaxed. It has a light yellowish-brown tint, or, as it is called, a faded-leaf color. Its consistence is reduced and the substance tears easily. In the left ventricle the papillary columns and the muscle beneath the endocardium show a streaked or

patchy appearance. Microscopically, the fibres are seen to be occupied by minute globules distributed in rows along the line of the primitive fibres (Welch). In advanced grades the fibres seem completely occupied by the minute globules.

(*b*) *Fatty Overgrowth.*—This is usually a simple excess of the normal subpericardial fat, to which the term *cor adiposum* was given by the older writers. In other instances the fat infiltrates the muscular substance and, separating the strands, may reach even to the endocardium. In corpulent persons there is always much pericardial fat. It forms part of the general obesity, and occasionally leads to dangerous or even fatal impairment of the contractile power of the heart. Of 122 cases analyzed by Forchheimer there were 88 males and 34 females. Over eighty per cent occurred between the fortieth and seventieth years.

The entire heart may be enveloped in a thick sheeting of fat through which not a trace of muscle substance can be seen. On section, the fat infiltrates the muscle, separating the fibres, and in extreme cases—particularly in the right ventricle—reaches the endocardium. In some places there may be even complete substitution of fat for the muscle substance. In rare instances the fat may be in the papillary muscles. The heart is usually much relaxed and the chambers are dilated. Microscopically the muscle fibres may show, in addition to the atrophy, marked fatty degeneration.

5. **Other Degenerations of the Myocardium.** (*a*) *Brown Atrophy.*—This is a common change in the heart-muscle, particularly in chronic valvular lesions and in the senile heart. When advanced, the color of the muscles is a dark red-brown, and the consistence is usually increased. The fibres present an accumulation of yellow-brown pigment chiefly about the nuclei. The cement substance is often unusually distinct, but seems more fragile than in healthy muscle.

(*b*) Amyloid degeneration of the heart is occasionally seen. It occurs in the intermuscular connective tissue and in the blood-vessels, not in the fibres.

(*c*) The hyaline transformation of Zenker is sometimes met with in prolonged fevers. The affected fibres are swollen, homogeneous, translucent, and the striæ are very faint or entirely absent.

(*d*) Calcareous degeneration may occur in the myocardium, and the muscle fibres may be infiltrated and yet retain their appearance as figured and described by Coats in his Text-book of Pathology.

Symptoms of Myocardial Disease.—These are notoriously uncertain. A man with advanced fibroid myocarditis may drop dead suddenly, while doing heavy work, without having complained of cardiac distress. On the other hand, a patient may present enfeebled, irregular action and signs of dilatation; he may have shortness of breath, œdema, and the general symptoms believed to be characteristic of cases of fibroid and fatty heart, and the post-mortem show little or no change in the myocardium.

Cardio-sclerosis or fibroid heart is in some cases characterized by a

feeble, irregular, slow pulse, with dyspnœa on exertion and occasional attacks of angina. Irregularity is present in many, but not in all cases. The pulse may be very slow, even 30 or 40 per minute. Ultimately the cases come under observation with the symptoms of cardiac insufficiency. The arrhythmia, which may have been present, becomes aggravated and, according to Riegel, may not only precede, but also persist after the cardiac insufficiency has passed away. This certainly does not hold in all cases, for a patient recently under observation had the most marked arrhythmia, which persisted after recovery from a severe attack of cardiac insufficiency in which he nearly died. Upon his return, a few weeks ago, with dilatation and arrhythmia, we agreed that the condition was probably one of cardio-sclerosis; but the autopsy showed simple dilatation without either fibroid or marked fatty change in the heart.

Fatty degeneration of the heart presents the same difficulties. Extreme fatty changes, as in pernicious anæmia, may be consistent with full, regular pulse and a regularly acting heart. In some of these cases the fat does not appear to interfere seriously with the function of the organ. The truth is it may exist in an extreme grade without producing symptoms, so long as great dilatation of the chambers does not occur. The cardiac irregularity, the dyspnœa, palpitation, and small pulse are in reality not symptoms of the fatty degeneration, but of dilatation which has supervened. The fatty *arcus senilis* is of no moment in the diagnosis of fatty heart. The heart-sounds may be weak and the action irregular. When dilatation occurs, there is often the gallop rhythm, shortening of the long pause, and a systolic murmur at the apex. Shortness of breath on exertion is an early feature in many cases, and anginal attacks may occur. There is sometimes a tendency to syncope, and in both fibroid and fatty heart there are attacks in which the patient feels cold and depressed and the pulse sinks to 40 or 30, or even, as in one case which I saw, to 26. The patient may wake from sleep in the early morning with an attack of severe cardiac asthma. These "spells" may be associated with nausea and may alternate with others in which there are anginal symptoms. These are the cases, too, in which for weeks there may be mental symptoms. The patient has delusions and may even become maniacal. Toward the close, the Cheyne-Stokes ~~breathing is met with in a number of cases~~.

Fatty overgrowth of the heart is a condition certain to exist in very obese persons. It produces no symptoms until the muscular fibre is so weakened that dilatation occurs. These patients may for years present a feeble but regular pulse; the heart-sounds are weak and muffled, and a murmur may be heard at the apex. Attacks of cardiac asthma are not uncommon, and the patient may suffer from bronchitis. Dizziness and pseudo-apoplectic seizures may occur. Sudden death may result from syncope or from rupture of the heart. The physical examination is often difficult because of the great increase in the fat, and it may be impossible to define the area of dulness.

and as those described by John Cheyne in a case of fatty heart-
(Dublin Hospital Reports vol II, p 221. 1818) "for several days his
breathing was irregular, it would entirely cease for a
quarter of a minute, then it would become perceptible, though
very low, then by degrees it became heaving & quick, and
then it would gradually cease again: this revolution in the
state of his breathing lasted about a minute, during which there
were about thirty acts of respiration

For practical purposes we may group the cases of myocardial disease as follows:

(1) Those in which sudden death occurs with or without previous indications of heart-trouble. Sclerosis of the coronary arteries exists—in some instances with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. ~~In~~ Many ~~cases there is~~ patient never ~~any~~ complain of cardiac distress, but, as in the case of Chalmers, the celebrated Scottish divine, ~~may~~ enjoy unusual vigor of mind and body.

(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms. These are the cases in which the condition may be strongly suspected and, in some instances, diagnosed. It is rarely possible to make a distinction between the fatty and fibroid heart.

(3) Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart. Dropsy is often present, and with a loud murmur at the apex it may be difficult, unless the case has been seen from the outset, to determine whether or not a valvular lesion is present.

Prognosis.—The outlook in affections of the myocardium is extremely grave. Patients recover, however, in a surprising way from the most serious attacks, particularly those of the second group.

Treatment.—Many cases never come under treatment; the first are the final symptoms.

Cases with signs of well-marked cardiac insufficiency, as manifested by dyspnœa, weak, irregular, rapid heart, and œdema, may be treated on the plan laid down for the treatment of broken compensation in valvular disease. Digitalis may be given even if fatty degeneration is suspected, and is often very beneficial.

Much more difficult is the management of those cases in which there is marked cardiac arrhythmia, with a feeble, irregular, very slow pulse, and syncope or angina. Dropsy is not, as a rule, present; the heart-sounds may be perfectly clear, and there are no signs of dilatation. Digitalis, under these circumstances, is not advisable, particularly when the pulse is infrequent. Complete rest in bed, a carefully regulated diet, and the use of the aromatic spirits of ammonia, sulphuric ether, and stimulants are indicated. For the restlessness and distressing feelings of anxiety morphia is invaluable. From an eightieth to a sixtieth of a grain of strychnia may be given three times a day. If, as is sometimes the case, the pulse is hard and firm, nitroglycerin may be cautiously administered, beginning with one minim of the one per cent solution three times a day and increased gradually.

In certain cases of weak heart, particularly when it is due to fatty overgrowth, the plan of treatment recommended by Oertl is advantageous. It is an invaluable method in those forms of heart-weakness due to intem-

perance in eating and drinking and defective bodily exercise. The Oertl plan consists of three parts. First, the reduction in the amount of liquid. This is an important factor in reducing the fat in these patients. It also slightly increases the density of the blood. Oertl allows daily about thirty-six ounces of liquid, which includes the amount taken with the solid food. Free perspiration is promoted by bathing (if advisable, the Turkish bath), or even by the use of pilocarpine.

The second important point in his treatment is the diet, which should consist largely of proteids.

Morning.—Cup of coffee or tea, with a little milk, about six ounces altogether. Bread, three ounces.

Noon.—Three to four ounces of soup, seven to eight ounces of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; one ounce of bread or farinaceous pudding; three to six ounces of fruit for dessert. No liquids at this meal, as a rule, but in hot weather six ounces of light wine may be taken.

Afternoon.—Six ounces of coffee or tea, with as much water. As an indulgence an ounce of bread.

Evening.—One or two soft-boiled eggs, an ounce of bread, perhaps a small slice of cheese, salad, and fruit; six to eight ounces of wine with four or five ounces of water (Yeo).

The most important element of all is graduated exercise, not on the level, but up hills of various grades. The distance walked each day is marked off and is gradually lengthened. In this way the heart is systematically exercised and strengthened.

There is no doubt of the great value of this or like methods in appropriate cases. At several of the health resorts in Germany, particularly the Bad Nauheim, under Schott, the results are striking. The plan is rarely advisable in valvular lesions and should not be adopted when there is marked arterio-sclerosis. Cases of fatty overgrowth of the heart are those most suitable. The plan of treatment reduces the obesity, and the patients are, for a time at least, much more comfortable and are able to go about and do their work without cardiac distress or great shortness of breath.

Aneurism of the Heart.

(*a*) **Aneurism of a Valve** results from acute endocarditis, which produces softening or erosion and may lead either to perforation of the segment or to gradual dilatation of a limited area under the influence of the blood-pressure. The aneurisms are usually spheroidal and project from the ventricular face of a sigmoid valve. They are much less common on the mitral segments. They frequently rupture and produce extensive destruction and incompetency of the valves.

(*b*) **Aneurism of the Walls.**—This comparatively rare condition results from the weakening of the walls by chronic myocarditis, or occasionally

it follows mural endocarditis, which more commonly, however, leads to perforation. Aneurism has followed a stab-wound of the heart. The left ventricle near the apex is usually the seat, at the situation in which the fibrous degeneration is most common. Fifty-nine of the 90 cases collected by Legg were situated here. In the early stages the anterior wall of the ventricle, near the septum, sometimes involving the septum itself, is slightly dilated, the endocardium opaque, and the muscular tissue sclerotic. In a more advanced stage the dilatation is pronounced and layers of thrombi occupy the sac. Ultimately a large rounded tumor may project from the ventricle and may attain a size equal to that of the heart. Occasionally the aneurism is sacculated and communicates with the ventricle through a very small orifice. The sac may be double, as in a case reported by Janeway. In the museum of Guy's Hospital there is a specimen showing the wall of the ventricle covered with aneurismal bulgings. Rupture occurred in 7 of the 90 cases collected by Legg.

The *symptoms* produced by aneurism of the heart are indefinite. Occasionally there is marked bulging in the apex region and the tumor may perforate the chest wall. When the sac is large and produces pressure upon the heart itself, there may be a marked disproportion between the strong cardiac impulse and the feeble pulsation in the peripheral arteries.

Rupture of the Heart.

This rare event is usually associated with fatty infiltration or degeneration of the heart-muscles. In some instances, acute softening in consequence of embolism of a branch of the coronary artery, suppurative myocarditis, or a gummatous growth has been the cause. Of 100 cases collected by Quain, fatty degeneration was noted in 77. Two thirds of the patients were over sixty years of age.

The rent may occur in any of the chambers, but is found most frequently in the left ventricle on the anterior wall, not far from the septum. The accident usually takes place during exertion. There may be no preliminary symptoms, but without any warning the patient may fall and die in a few moments. Sudden death occurred in seventy-one per cent of Quain's cases. In other instances there may be in the cardiac region a sense of anguish and suffocation, and life may be prolonged for several hours. In a Montreal case which I examined the patient walked up a steep hill after the onset of the symptoms, and lived for thirteen hours. A case is on record in which the patient lived for eleven days.

New Growths and Parasites.

Tubercle and syphilis have already been considered. Primary cancer or sarcoma is extremely rare. Secondary tumors may be single or multiple, and are usually unattended with symptoms, even when the disease

is most extensive. In one case I found in the wall of the right ventricle a mass which involved the anterior segment of the tricuspid valve and partly blocked the orifice. The surface was eroded and there were numerous cancerous emboli in the pulmonary artery. In another instance the heart was greatly enlarged, owing to the presence of innumerable masses of colloid cancer the size of cherries. The mediastinal sarcoma may penetrate the heart, though it is remarkable how extensive the disease of the mediastinal glands may be without involvement of the heart or vessels.

Cysts in the heart are rare. They are found in different parts, and are filled either with a brownish or a clear fluid. Blood-cysts occasionally occur.

The parasites will be discussed under the appropriate section, but it may be mentioned here that both the *cysticercus cellulosæ* and the echinococcus cysts occur occasionally in the heart.

Wounds and Foreign Bodies.

Wounds of the heart are usually fatal, although there are many instances in which recovery has taken place. Bullets have been found encysted inside the ventricle. A majority of the cases of gunshot wounds, however, are necessarily fatal. Puncture of the heart by a sharp-pointed body, such as a needle or a stiletto, does not always prove fatal. Peabody has reported a case in which a pin was found embedded in the left ventricle. Suicide has been attempted by passing a needle or pin into the heart. It is not, however, necessarily fatal. Moxon mentioned a case, at the Clinical Society of London, in which a medical student, while on a spree, passed a pin into his heart. The pericardium was opened, and the head of the pin was found outside of the right ventricle. It was grasped and an attempt made to remove it, but it was withdrawn into the heart and, it is said, caused the patient no further trouble. Hysterical girls sometimes swallow pins and needles, which, passing through the œsophagus and stomach, are found in various parts of the body. A remarkable case is reported by Allen J. Smith of a girl from whom several dozen needles and pins were removed, usually from subcutaneous abscesses. Several years later she developed symptoms of chronic heart-disease. At the post-mortem needles were found in the tissues of the adherent pericardium, and between thirty and forty were embedded in the thickened pleural membranes of the left side.

Puncture of the heart has been recommended as a therapeutic procedure to stimulate it to action, as in chloroform narcosis, and experimental evidence has been brought forward by B. A. Watson in favor of the operation. He advises abstraction of blood in combination with the puncture—cardiocentesis. The proceeding is not without risk. Hæmorrhage may take place from the puncture, though it is not often extensive.

At the Philadelphia Hospital the procedure was tried by one of the resident physicians in a case of acute dilatation. The anterior coronary vein was cut across and considerable blood was found in the pericardium. There is danger also of striking Kronecker's inhibition centre.

V. NEUROSES OF THE HEART.

Palpitation.

In health we are unconscious of the action of the heart. In some people one of the first indications of debility or overwork is the consciousness of the cardiac pulsations, which may, however, be perfectly regular and orderly. This is not palpitation. The term is properly limited to irregular or forcible action of the heart perceptible to the individual.

Etiology.—The expression "perceptible to the individual" covers the essential element in palpitation of the heart. The most extreme disturbance of rhythm, a condition even of what is termed *delirium cordis*, may be unattended with subjective sensations of distress, and there may be no consciousness of disturbed action. On the other hand, there are cases in which complaint is made of the most distressing palpitation and sensations of throbbing, in which the physical examination reveals a regularly acting heart, the sensations being entirely subjective. We meet with this symptom in a large group of cases in which there is increased excitability of the nervous system. Palpitation may be a marked feature at the time of puberty, at the climacteric, and occasionally during menstruation. It is a very common symptom in hysteria and neurasthenia, particularly in the form of the latter which is associated with dyspepsia. Emotions, such as fright, are common causes of palpitation. It may occur as a sequence of the acute fevers. Females are more liable to the affection than males.

In a second group the palpitation results from the action upon the heart of certain substances, such as tobacco, coffee, tea, and alcohol. And, lastly, palpitation may be associated with organic disease of the heart, either of the myocardium or of the valves. As a rule, however, it is a purely nervous phenomenon—seldom associated with organic disease—in which the most violent action and the most extreme irregularity may exist without that subjective element of consciousness of the disturbance which constitutes the essential feature of palpitation.

The irritable heart described by Da Costa, which was so common among the young soldiers during the civil war, is a neurosis of this kind. The chief symptoms were palpitation with great frequency of the pulse on exertion, a variable amount of cardiac pain, and dyspnœa. The factors at work in producing this condition appeared to be the mental excitement, the unwonted muscular exertion associated with the drill, and diarrhœa.

The condition is not infrequent in civil life among young men, and it leads in some cases to hypertrophy of the heart.

Symptoms.—In the mildest form, such as occurs during a dyspeptic attack, there is slight fluttering of the heart and a sense of what patients sometimes call "goneness." In more severe attacks the heart beats violently, its pulsations against the chest wall are visible, the rapidity of the action is much increased, the arteries throb forcibly, and there is a sense of great distress. In some instances the heart's action is not at all quickened. The most striking cases are in neurasthenic women, in whom the mere entrance of a person into the room will cause the most violent action of the heart and throbbing of the peripheral arteries. The pulse may be rapidly increased until it reaches 150 or 160. A diffuse flushing of the skin may appear at the same time. After such attacks, there may be the passage of a large quantity of pale urine. In many cases of palpitation, particularly in young men, the condition is at once relieved by exertion. A patient with extreme irregularity of the heart may, after walking quickly one hundred yards or running up-stairs, return with the pulse perfectly regular. This is not infrequently seen, too, in the irregular action of the heart in mitral-valve disease.

The physical examination of the heart is usually negative. The sounds, the shock of which may be very palpable, are on auscultation clear, ringing, and metallic, but not associated with murmurs. The second sound at the base may be greatly accentuated. A murmur may sometimes be heard over the pulmonary artery or even at the apex in cases of rapid action in neurasthenia or in severe anæmia. The attacks may be transient, lasting only for a few minutes, or may persist for an hour or more. In some instances any attempt at exertion renews the attack.

The *prognosis* is usually good, though it may be extremely difficult to remove the conditions underlying the palpitation.

Arrhythmia.

An intermission occurs when one or more beats of the heart are dropped. Irregularity is the condition when the beats are unequal in volume and force, or follow each other at unequal distances. Allorrhythmia is a term which is also used to express deviations from the normal heart rhythm.

The following varieties of arrhythmical action may be recognized:

(1) The paradoxical pulse of Küssmaul, in which the beats during inspiration are more frequent but less full than during expiration. This is found in weak heart, in chronic pericarditis, and when fibrous bands encircle the root of the aorta; but it may also occur normally from the influence of the respirations upon the heart. It is sometimes to be felt in sleeping children.

(2) Intermittence, in which there is simply an intermission or drop-

and as an effect of Digitalis

ping of a cardiac beat. The term deficience is more correctly applied to those instances in which the absence of the heart-sound proves that the systole is really omitted. The systole may be so weak as not to produce a pulsation, and yet at the same time a feeble first sound may be heard.

(3) The alternate heart-beat, in which strong and weak pulsations alternate regularly and which is expressed in the peripheral arteries by alternate full and feeble pulse-beats.

(4) The bigeminal and trigeminal pulsations occur when two or three beats follow each other in rapid succession, each group being separated from the following by a longer interval. This is not very uncommon in mitral disease. In the bigeminal pulse the first beat of the pair is usually the stronger. Indeed, in the condition known as heart bigeminism the second systole is so feeble that the pulse wave does not reach the peripheral arteries and the two systoles are represented by only a single pulse-beat at the wrist.

(5) Delirium cordis, in which these various factors are combined and the heart's action is wholly irregular.

(6) Fœtal heart rhythm—embryocardia—described by Stokes, is a very common condition in which the long pause is shortened and the characters of the sounds are "almost completely identical." The resemblance to the fœtal heart-beat is very striking. In the later stages of fevers and in extreme dilatation this form of heart rhythm is very frequently heard.

(7) Gallop rhythm, in which the sounds resemble the footfall of a horse at canter, usually results from the reduplication of the second sound in a rapidly acting heart. It is expressed by the words "rat-ta-tat." Sometimes it seems as if the first sound was split; more commonly it is the second. It is most frequently heard in interstitial nephritis and arterio-sclerosis, but it is said to be met with also in healthy persons.

The causes of these various disturbances of rhythm are thus classified by G. Baumgarten: *

(1) Those due to central—cerebral—causes, either organic disease, as in hæmorrhage, or concussion; more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of arrhythmia. Various drugs, such as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort and may have an important influence in producing disturbances in the rhythm; but as yet we do not know their exact significance. They

* Transactions of the Association of American Physicians, vol. iii.

may be present in cases which have not presented arrhythmia. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter usually associated with sclerosis of the coronary arteries.

The significance of arrhythmia is not always easy to determine. Simple irregular action of the heart may persist for years. The late Chancellor Ferrier, of McGill University, a man of unusual bodily and mental vigor, who died at the age of eighty-seven, had an extremely irregular pulse for almost fifty years of his life. One or two other instances have come under my notice of persons in good health, without arterial or cardiac disease, in whom the heart's action was persistently irregular. The bigeminal and trigeminal pulsations are found more frequently in mitral than in other conditions. The delirium cordis is met with in the dilatation associated with valvular lesions, particularly toward the latter stages. Fœtal heart rhythm is rarely found apart from dilatation.

Rapid Heart—Tachycardia.

The rapid action may be perfectly natural. There are individuals whose normal heart action is at 100 or even more per minute. It may be caused by the various conditions which induce palpitation; but the two are not necessarily associated. Emotional causes, violent exercise, and fevers all produce great increase in the rapidity of the heart's action. The extremely rapid action which follows fright may persist for days, or even weeks. Traube reports an instance in which, after violent exercise, the rapid action of the heart continued. Cases are not uncommon at the menopause.

There are cases again in which the condition can hardly be termed a neurosis, since it depends upon definite changes in the pneumogastrics or in the medulla. Cases have been reported in which tumor or clot in or about the medulla or pressure upon the vagi has been associated with heart hurry. Some of the cases of frequent action of the heart in women have been thought to be due to reflex irritation from ovarian or uterine disease.

Paroxysmal tachycardia is a remarkable affection, characterized by spells of heart hurry, during which the action is greatly increased, the pulse reaching 200 and over. The cases are not common. The condition has been thoroughly studied by Nothnagel. The attack may be quite short and persist only for an hour or so. A patient at the Philadelphia Infirmary for Nervous Diseases was attacked every week or two; the pulse would rise to 220 or 230, and there were such feelings of distress and uneasiness that the patient always had to lie down. There may be, however, no subjective disturbance, and in another case the patient was able to walk about during the paroxysm and had no dyspnœa. One of the most remarkable cases is reported by H. C. Wood. A physician in his eighty-

seventh year has had attacks at intervals since his thirty-seventh year. The onset is abrupt and the pulse rapidly rises to 200 a minute. For more than twenty years the taking of ice-water or strong coffee would arrest the attacks. Bouveret has analyzed a number of cases of this essential or idiopathic form; he finds that a permanent cure is rare, and that the patients suffer for ten or more years. Four instances terminated fatally from heart-failure. Wood suggests that these cardiac paroxysms are caused by discharging lesions affecting the centres of the accelerator nerves. François Franck has shown that the acceleration of the heart's action is due to the shortening of the diastole, and during the systole so little blood is expelled from the heart that the average amount in the minute is not increased. Moreover, the accelerators appear to have no trophic relation to the heart, and stimulation of them is not accompanied either by increased arterial pressure or by augmentation of the work done by the heart.

Slow Heart—Brachycardia (*Bradycardia*).

Slow action of the heart is sometimes normal and may be a family peculiarity. Napoleon is stated to have had a pulse of only 40 per minute.

In any case of slow pulse it is important first to make sure that the number of heart and arterial beats correspond. In many instances this is not the case, and with a radial pulse at 40 the cardiac pulsations may be 80, half the beats not reaching the wrist. The heart contractions, not the pulse wave, should be taken into account. A most exhaustive study of this condition has been made recently by Riegel, whose division is here followed:

(*a*) Physiological brachycardia. In the puerperal state the pulse may beat from 44 to 60 per minute, or may even be as low as 34. It is seen in premature labor as well as at term. The explanation of its occurrence at this period is not clear. Slowness of the pulse is associated with hunger. Brachycardia depending on individual peculiarity is extremely rare.

(*b*) Pathological brachycardia, which is met with under the following conditions: (1) In convalescence from acute fevers. This is extremely common, particularly after pneumonia, typhoid fever, acute rheumatism, and diphtheria. It is most frequently seen in young persons and in cases which have run a normal course. Traube's explanation that it is due to exhaustion is probably the correct one. (2) In diseases of the digestive system, such as chronic dyspepsia, ulcer or cancer of the stomach, and jaundice. The largest number of Riegel's cases were of this group. (3) In diseases of the respiratory system. Here it is by no means so common, but is seen not infrequently in emphysema. (4) In diseases of the circulatory system. Excluding all cases of irregularity of the heart, brachycardia is not common in diseases of the valves. It is most frequently seen in fatty and fibroid changes in the heart, but is not constant in them. (5) In diseases of the urinary organs. It occurs occasionally in nephritis and

may be a feature of uræmia. (6) From the action of toxic agents. It occurs in uræmia, poisoning by lead, alcohol, and follows the use of tobacco, coffee, and digitalis. (7) In constitutional disorders, such as anæmia, chlorosis, and diabetes. (8) In diseases of the nervous system. Apoplexy, epilepsy, the cerebral tumors, affections of the medulla, and diseases and injuries of the cervical cord may be associated with very slow pulse. In general paresis, mania, and melancholia it is not infrequent. (9) It occurs occasionally in affections of the skin and sexual organs, and in sunstroke, or in prolonged exhaustion from any cause.

It is seen most frequently in the convalescence from acute fevers, then in disorders of the digestive system. The significance of this symptom is variable. It is only in diseases of the heart or brain that it is ominous. It may be due to direct irritation of the vagi, to diminished excitability of the cardiac ganglia, to reflex influences acting upon the vagus centre, or to weakness of the heart-muscle itself. The pulse-beat rarely sinks below 20. Prentice, at the Association of American Physicians at Washington, showed a patient with attacks of unconsciousness, who had, particularly during the attacks, but also in the intervals, a pulse as low as 12 per minute. Such cases are extremely rare. Cases are on record in which the pulse has fallen to 8 or 9 beats in the minute. At the discussion which followed the exhibition of Prentice's patient, both Jacobi and Kinnicutt referred to similar cases associated with epileptic seizures, in one of which the pulse fell as low as 7 in the minute.

Treatment of Palpitation and Arrhythmia.—An important element in many cases is to get the patient's mind quieted, and he can be assured that there is no actual danger. The mental element is oftentimes very strong. In palpitation, before using medicines, it is well to try the effect of hygienic measures. As a rule, moderate exercise may be taken with advantage. Regular hours should be kept, and at least ten hours out of the twenty-four should be spent in the recumbent posture. A tepid bath may be taken in the morning, or, if the patient is weakly and nervous, in the evening, followed by a thorough rubbing. Hot baths and the Turkish bath should be avoided. The dietetic management is most important. It is best to prohibit absolutely alcohol, tea, and coffee. The diet should be light and the patient should avoid taking large meals. Articles of food known to cause flatulency should not be used. If a smoker, the patient should give up tobacco. Sexual excitement is particularly pernicious, and the patient should be warned specially on this point. For the distressing attacks of palpitation which occur with neurasthenia, particularly in women, a rigid Weir-Mitchell course is the most satisfactory. It is in these cases that we find the most distressing throbbing in the abdomen, which is apt to come on after meals, and is very much aggravated by flatulency. The cases of palpitation due to excesses or to errors in diet and dyspepsia are readily remedied by hygienic measures.

See Jenner's letter about Hunter in
Baron's Life of Jenner

A course of iron is often useful. Strychnia is particularly valuable, and is perhaps best administered as the tincture of nux vomica in large doses. Very little good is obtained from the smaller quantities. It should be given freely, 20 minims three times a day.

If there is great rapidity of action, aconite may be tried or veratrum viride. There are cases associated with sleeplessness and restlessness which are greatly benefited by bromide of potassium. Digitalis is very rarely indicated, but in obstinate cases it may be tried with the nux vomica.

Cases of heart hurry are often extremely obstinate, as may be judged from the case of the physician reported by H. C. Wood, in whom the condition persisted in spite of all measures for fifty years. The bromides are sometimes useful; the general condition of neurasthenia should be treated, and during the paroxysm an ice-bag may be placed upon the heart, or Leiter's coil, through which ice-water may be passed. Electricity, in the form of galvanism, is sometimes serviceable, and for its mental effect the Franklinic current. For the condition of slow pulse but little can be done. A great majority of the cases are not dangerous.

Angina Pectoris.

Stenocardia, or the breast-pang described by Heberden, is not an independent affection, but a symptom associated with a number of morbid conditions of the heart and vessels, more particularly with sclerosis of the root of the aorta and changes in the coronary arteries. True angina, which is a rare disease, is characterized by paroxysms of agonizing pain in the region of the heart, extending into the arms and neck. In violent attacks there is a sensation of impending death.

Etiology and Pathology.—It is a disease of adult life and occurs almost exclusively in men. Arterio-sclerosis, hypertrophy of the heart, increased arterial tension, or aortic insufficiency are often present, while anatomical changes in the aorta, arteries, and myocardium are almost constant. No instance of true angina has come under my observation in which there were not signs of cardio-vascular changes. The immediate exciting cause of an attack is most frequently sudden exertion or emotional excitement. The paroxysm may come on in the daytime, but in some of the worst cases they occur at night. The nature of the affection is doubtful. The following views have been entertained.

(1) That it is a neuralgia of the cardiac nerves. In the true form the agonizing cramp-like character of the pain, the suddenness of the onset, and the associated features, are unlike any neuralgic affection. The pain, however, is undoubtedly in the cardiac plexus and radiates to adjacent nerves. It is interesting to note in connection with the almost constant sclerosis of the coronary arteries in angina that Thoma has found marked sclerosis of the temporal artery in migraine and Dana has met with local thickening of the arteries in some cases of neuralgia (2) Heb-

erden believed that it was a cramp of the heart-muscle itself. This would explain the agonizing character of the pain and the suddenness of the onset as well as the frequency of the fatal termination; but if the cramps were general in the heart-muscle and similar to those which occur in the voluntary muscles, death would invariably ensue with great rapidity. Cramp of certain muscular territories would explain the attack. (3) That it is due to the extreme tension of the ventricular walls, in consequence of an acute dilatation associated, in the majority of cases, with affection of the coronary arteries. Traube, who supported this view, held that the agonizing pain resulted from the great stretching and tension of the nerves in the muscular substance. A modified form of this view is that there is a spasm of the coronary arteries with great increase of the intracardiac pressure.

In fatal cases of angina the coronary arteries are almost invariably diseased, either in their main division, or there is chronic endarteritis with great narrowing of the orifices at the root of the aorta. Experimentally, occlusion of the coronary arteries produces slowing of the heart's action, gradual dilatation, and death within a very few minutes. Cohnheim has shown that in the dog ligation of one of the large coronary branches produces within a minute a condition of arrhythmia, and within two minutes the heart ceases in diastole. These experiments, however, do not throw much light upon the etiology of angina pectoris. Extreme sclerosis of the coronary arteries is common, and a large majority of the cases present no symptoms of angina. Even in the cases of sudden death due to blocking of an artery, particularly the anterior branch of the coronary artery, there is usually no great pain either before or during the attack. The lesions of the nerves described by Lancereaux, Hadden, and others cannot yet be correlated satisfactorily with the symptoms of true angina. Various forms of true angina have been recognized, but the differences, in the majority of instances, are not sufficiently marked to permit a separation. Reference may be made, however, to the angina pectoris *vaso-motoria* described by Nothnagel. In this the attack may come on after exposure to cold. There is general spasm of the peripheral arteries with a sense of stiffness and deadness in the extremities, and pallor, cyanosis, and lowering of the temperature. The arteries are small and contracted. There is sometimes a feeling of faintness or even a loss of consciousness. With this there is a sense of pressure, tension, or even agonizing pain in the cardiac region. The pulse, however, is regular, and there are no signs of disease of the heart. The condition is supposed to depend upon a wide-spread spasm of the peripheral arteries. I have never recognized a case of this kind, although certain of its features are not at all uncommon in the pseudo-angina.

Symptoms.—Usually during exertion or intense mental emotion the patient is seized with an agonizing pain in the region of the heart and a sense of constriction, as if the heart had been seized in a vise. The pains

(4) Potain's new — see Lukens paper

Poteris ether in legal of asthenics

Angina of the face & neck may however be present

radiate up the neck and down the arm and there may be numbness of the fingers or in the cardiac region. The face is usually pallid and may assume an ashy-gray tint, and not infrequently a profuse sweat breaks out over the surface. Dyspnœa is not usually present. The paroxysm lasts from several seconds to a minute or two, during which, in severe attacks, the patient feels as if death were imminent. There is great restlessness and anxiety, and the patient may drop dead at the height of the attack or faint and pass away in syncope. The condition of the heart during the attack is variable; the pulsations may be uniform and regular. The pulse tension, however, is usually increased, but it is surprising, even in cases of extreme severity, how slightly the character of the pulse may be altered. After the attack there may be eructations, or the passage of a large quantity of clear urine. The patient usually feels exhausted, and for a day or two may be badly shaken; in other instances in an hour or two the patient feels himself again. The attacks may recur at intervals of a few weeks, or perhaps not for many years. There are individuals who have well-marked anginal attacks for years, and, except during the paroxysms, suffer but slight inconvenience.

Diagnosis.—There are many grades of true angina. A man may have slight præcordial pain, a sense of distress and uneasiness, and radiation of the pains to the arm and neck. Such attacks following slight exertion, an indiscretion in diet, or a disturbing emotion may alternate with attacks of much greater severity, or they may occur in connection with a pulse of increased tension and signs of general arterio-sclerosis. In the milder grades the diagnosis cannot rest upon the symptoms of the attack itself, since they may be simulated by the pseudo-angina; but the diagnosis should be based upon the examination of the circulatory system. In true angina, even in the milder forms, signs of arterio-sclerosis are usually present. In a case presenting attacks of præcordial pain or pains in the cervical or brachial plexuses, if the aortic second sound is clear, not ringing, the pulse tension low, and the peripheral arteries soft, the diagnosis of true angina should not be made. After all, the chief difficulty, however, arises in the cases of the hysterical or *pseudo-angina.*

This is a common affection in women, but may occur also in neurasthenic men. It is in this form particularly that we see vaso-motor phenomena. The patient may complain of great coldness of the hands or feet, or a general feeling of deadness and stiffness, often with pain in the back of the head and neck. The attacks recur frequently, and sometimes become worse at each monthly period. They may come on with great severity at the menopause. Worry and disturbing emotions of all kinds may at any time precipitate an attack. Huchard has given in concise form the following points in diagnosis between the true and hysterical angina:

TRUE ANGINA.	PSEUDO-ANGINA.
Most common between the ages of forty and fifty years.	At every age, even six years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely periodical or nocturnal.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with nervous symptoms.
Vaso-motor form rare. Agonizing pain and sensation of compression by a vise.	Vaso-motor form common. Pain less severe; sensation of distention.
Pain of short duration. Attitude: silence, immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions: sclerosis of coronary artery.	Neuralgia of nerves and cardio-plexus.
Prognosis grave, often fatal.	Never fatal.
Arterial medication.	Antineuralgic medication.

There are cases in women which are sometimes very puzzling; for instance, when the patient presents a combination of marked hysterical manifestations and attacks of angina and has aortic insufficiency. In such instances the patient should receive the benefit of the doubt and be treated for true angina.

Prognosis.—Cardiac pain without evidence of arterio-sclerosis or valve disease is not of much moment. True angina is almost invariably associated with marked cardio-vascular lesions in which the prognosis is always grave. With judicious treatment the attacks, however, may be long deferred, and a few instances recover completely. The prognosis is naturally more serious with aortic insufficiency and advanced arterio-sclerosis. Patients who have had well-marked attacks may live for many years, but much depends upon the care with which they regulate their daily life.

Treatment.—Patients subject to this affection should live a quiet life, avoiding particularly excitement and sudden muscular exertion. During the attack nitrite of amyl should be inhaled, as advised by Lauder Brunton. From two to five drops may be placed upon cotton-wool in a tumbler or upon the handkerchief. This is frequently of great service in the attack, relieving the agonizing pain and distress. Subjects of the disease should carry the *perles* of the nitrite of amyl with them, and use them on the first indication of an attack. In some instances the nitrite of amyl is quite powerless, though given freely. If within a minute or two relief is not obtained in this way, chloroform should at once be given. A few inhalations act promptly and give great relief. Should the pains continue, a hypodermic of morphia may be administered.

Saline Purgatives should be given

It is true be remembers that in true angina the patient may resist the influence of Morphia in a remarkable manner. In a case where I saw with Dr Poole grs ½ of morphia were given within twelve hours!

In the intervals, nitroglycerin may be given in full doses, as recommended by Murrell, or the nitrite of sodium (Matthew Hay). The nitroglycerin should be used for a long time and in increasing doses, beginning with one minim three times a day of the one per cent solution, and increasing the dose one minim every five or six days until the patient complains of flushing or headache.

Huchard recommends the iodides, believing that their prolonged use influences the arterio-sclerosis. Twenty grains three times a day may be given for several years, omitting the medicine for about ten days in each month. In some instances this treatment is certainly beneficial. Two men, both with arterio-sclerosis, ringing, accentuated aortic sound, and attacks of true angina, have under its use remained practically free from attacks—one case for nearly three, and the other for fully four years. This treatment is, however, not always satisfactory, and I have had several cases in which the condition has not been at all relieved by it.

For the pseudo-angina, the treatment must be directed to the general nervous condition. Electricity is sometimes very beneficial, particularly the Franklinic form.

VI. CONGENITAL AFFECTIONS OF THE HEART.

These have only a limited clinical interest, as in a large proportion of the cases the anomaly is not compatible with life, and in others nothing can be done to remedy the defect or even to relieve the symptoms.

The congenital affections result from interruption of the normal course of development or from inflammatory processes—endocarditis; sometimes from a combination of both.

(*a*) Of *general anomalies* of development the following conditions may be mentioned: *Acardia*, absence of the heart, which has been met with in the monstrosity known by the same name; *double heart*, which has occasionally been found in extreme grades of fœtal deformity; *dextrocardia*, in which the heart is on the right side, either alone or as part of a general transposition of the viscera; *ectopia cordis*, a condition associated with fission of the chest wall and of the abdomen. The heart may be situated in the cervical, pectoral, or abdominal regions. Except in the abdominal variety the condition is very rarely compatible with extra-uterine life.

(*b*) **Anomalies of the Cardiac Septa.**—The septa of both auricles and ventricles may be defective, in which case the heart consists of but two chambers, the *cor biloculare* or reptilian heart. In the septum of the auricles there is a very common defect, owing to the fact that the membrane closing the foramen ovale has failed at one point to become attached to the ring, and leaves a valvular slit which may be large enough to admit the

handle of a scalpel. Neither this nor the small cribriform perforations of the membrane are of any significance.

The foramen ovale may be patent without a trace of membrane closing it. In some instances this exists with other serious defects, such as stenosis of the pulmonary artery, or imperfection of the ventricular septum. In others the patent foramen ovale is the only anomaly, and in many instances it does not appear to have caused any embarrassment, as the condition has been found in persons who have died of various affections. The ventricular septum may be absent, the condition known as trilocular heart. Much more frequently there is a small defect in the upper portion of the septum, either in the situation of the membranous portion known as the "undefended space" or in the region situated just anterior to this. The anomaly is very frequently associated with narrowing of the pulmonary orifice or of the conus arteriosus of the right ventricle.

(*c*) **Anomalies and Lesions of the Valves.**—Numerical anomalies of the valve are not uncommon. The semilunar segments at the arterial orifices are not infrequently increased or diminished in number. Supernumerary segments are more frequent in the pulmonary artery than in the aorta. Four, or sometimes five, valves have been found. The segments may be of equal size, but, as a rule, the supernumerary valve is small.

Instead of three there may be only two semilunar valves, or, as it is termed, the *bicuspid condition.* In my experience, this is most frequent in the aortic valve. Of twenty-one instances only two occurred at the pulmonary orifice. Two of the valves have united, and from the ventricular face show either no trace of division or else a slight depression indicating where the union had occurred. From the aortic side there is usually to be seen some trace of division into two sinuses of Valsalva. There has been a discussion as to the origin of this condition, whether it is really an anomaly or whether it is not due to endocarditis, fœtal or post-natal. The combined segment is usually thickened, but the fact that this anomaly is met with in the fœtus without a trace of sclerosis or endocarditis shows that it may, in some cases at least, result from a developmental error.

Clinically this is a very important congenital defect, owing to the liability of the combined valve to sclerotic changes. Except two fœtal specimens all of my cases showed thickening and deformity, and in fifteen of those which I have reported death resulted directly or indirectly from the lesion.

The little fenestrations at the margins of the sigmoid valves have no significance; they occur in a considerable proportion of all bodies.

Anomalies of the auriculo-ventricular valves are not often met with.

Fœtal endocarditis may occur either at the arterial or auriculo-ventricular orifices. It is nearly always of the chronic or sclerotic variety. Very rarely indeed is it of the warty or verrucose form. There are little nodular bodies, sometimes six or eight in number, on the mitral and tricuspid segments—the nodules of Albini—which represent the remains of

Ductus Botalli – persistence of. septum ventr.

Hypertrophy of the right ventricle

fœtal structures, and must not be mistaken for endocardial outgrowths. The little rounded, bead-like hæmorrhages of a deep purple color, which are very common on the heart valves of children, are also not to be mistaken for the products of endocarditis. In fœtal endocarditis the segments are usually thickened at the edges, shrunken, and smooth. In the mitral and tricuspid valves the cusps are found united and the chordæ tendineæ are thickened and shortened. In the semilunar valves all trace of the segments has disappeared, leaving a stiff membranous diaphragm perforated by an oval or rounded orifice. It is sometimes very difficult to say whether this condition has resulted from fœtal endocarditis or whether it is an error in development. In very many instances the processes are combined; an anomalous valve becomes the seat of chronic sclerotic changes, and, according to Rauchfuss, endocarditis is more common on the right side of the heart only because the valves are here most often the seat of developmental errors.

Lesions at the Pulmonary Orifice.—*Stenosis* of this orifice is one of the commonest and most important of congenital heart affections. A slow endocarditis causes gradual union of the segments and narrowing of the orifice to such a degree that it only admits the smallest-sized probe. In some of the cases the smooth membranous condition of the combined segments is such that it would appear to be the result of faulty development. In some instances vegetations develop. The condition is compatible with life for many years, and in a considerable proportion of the cases of heart-disease above the tenth year this lesion is present. With it there may be defect of the ventricular septum. Obliteration or *atresia* of the pulmonary orifice is less frequent but a more serious condition than stenosis. It is of necessity associated with either imperfection of the ventricular septum or patency of the foramen ovale and persistence of the ductus arteriosus. *Stenosis of the conus arteriosus* of the right ventricle exists in a considerable proportion of the cases of obstruction at the pulmonary orifice. At the outset a developmental error, it may be combined with sclerotic changes. The ventricular septum is imperfect, the foramen ovale is usually open, and the ductus arteriosus patent. These three lesions at the pulmonary orifice constitute the most important group of all congenital cardiac affections. Of 181 instances of various congenital anomalies collected by Peacock 119 cases came under this category, and, according to this author, in eighty-six per cent of the patients with congenital heart-disease living beyond the twelfth year the lesion is at this orifice.

Congenital lesions of the aortic orifice are not very frequent. Rauchfuss has collected 24 cases of stenosis and atresia, and stenosis of the left conus arteriosus may also occur, a condition which is not incompatible with prolonged life. Ten of the sixteen cases tabulated by Dilg were over thirty years of age.

Symptoms of Congenital Heart-disease.—Cyanosis occurs in over ninety per cent of the cases and forms so distinctive a feature that

the terms "blue disease" and "morbus cæruleus" are practically synonyms for congenital heart-disease. The lividity in a majority of cases appears early, within the first week of life, and may be general or confined to the lips, nose, and ears, and to the fingers and toes. In some instances there is in addition a general dusky suffusion, and in the most extreme grades the skin is almost purple. It may vary a good deal and may only be intense on exertion. The external temperature is low. Dyspnœa on exertion and cough are common symptoms. The children rarely thrive and often display a lethargy of both mind and body. The fingers and toes are clubbed in a grade rarely met with in any other affection. The cause of the cyanosis has been much discussed. Morgagni referred it to the general congestion of the venous system due to obstruction, and this view was supported in a paper, one of the ablest that has been written on the subject, by Moreton Stillé. Morrison's recent analysis of 75 cases of congenital heart-disease shows that closure of the pulmonary orifice and patency of the foramen ovale and the ventricular septum are the lesions most frequently associated with cyanosis, and he concludes that the deficient aëration of the blood owing to diminished lung function is the most important factor. Another view, advocated by William Hunter, was that the discoloration was due to the admixture in the heart of venous and arterial blood; but lesions may exist which permit of very free mixture without producing cyanosis.

Diagnosis.—In the case of children, cyanosis, with or without enlargement of the heart, and the existence of a murmur are sufficient, as a rule, to determine the presence of a congenital heart-lesion. The cyanosis gives us no clew to the precise nature of the trouble, as it is a symptom common to many lesions and it may be absent in certain conditions. The murmur is usually systolic in character. It is, however, not always present, and there are instances on record of complicated congenital lesions in which the examination showed normal heart-sounds. In two or three instances fœtal endocarditis has been diagnosed *in gravida* by the presence of a rough systolic murmur, and the condition has been corroborated subsequent to the birth of the child. Hypertrophy is present in a majority of the cases of congenital defect. It is impossible in the scope of a work of this sort to enter upon elaborate details in differential diagnosis between the various congenital heart-lesions. I here abstract the conclusions on this question given by Hochsinger in his recent monograph:*

"(1) In childhood, loud, rough, musical heart-murmurs, with normal or only slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired endocardial defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness.

"(2) In young children heart-murmurs with great increase in the

* Die Auscultation des kindlichen Herzens, Wien, 1890.

cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(4) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and cannot be explained by the assumption of an endocarditis of the aortic valve."

Treatment.—The child should be warmly clad and guarded from all circumstances liable to excite bronchitis. In the attacks of urgent dyspnœa with lividity blood should be freely let. Saline cathartics are also useful. Digitalis must be used with care, and it is sometimes beneficial in the later stages. When the compensation fails, the indications for treatment are those of valvular disease in adults.

III. DISEASES OF THE ARTERIES.

I. DEGENERATIONS.

Fatty degeneration of the intima is extremely common, and is seen in the form of yellowish-white spots in the aorta and larger vessels. *Calcification* of the arterial wall follows fatty degeneration, atheromatous changes, and sclerosis. It occurs in either the intima or the media. In the latter it produces what is sometimes known as annular calcification, which occurs particularly in the middle coat of medium-sized vessels and may convert them into firm tubes. Calcification of the intima is a common terminal process of arterio-sclerosis.

Hyaline degeneration may attack either the larger or the smaller vessels. In the former the intima is converted into a smooth, homogeneous substance, and it is commonly an initial stage of arterio-sclerosis. In the smaller arteries and capillaries the hyaline degeneration is often seen, particularly in the glomeruli of the kidney. Its exact production is still a matter of some doubt. "It appears to arise principally by homogeneous coagulation of an albuminous fluid, either within the vessels or infiltrating the cells and the hyaline transformation of proliferating cells and of leucocytes."

II. ARTERIO-SCLEROSIS (*Arterio-capillary Fibrosis*).

The conception of arterio-sclerosis as an independent affection—a general disease of the vascular system—is due to Gull and Sutton.

Definition.—A condition of thickening, diffuse or circumscribed, of the intima, consequent upon primary changes in the media and adventitia. The process leads, in the larger arteries, to what is known as atheroma or endarteritis deformans.

Etiology.—(1) As an involution process arterio-sclerosis is an accompaniment of old age, and is the expression of the natural wear and tear to which the tubes are subjected. Longevity is a vascular question, and has been well expressed in the axiom that "a man is only as old as his arteries." To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arterio-sclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondly upon the amount of wear and tear to which he has subjected it. That the former plays the most important *rôle* is shown in the cases in which arterio-sclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Thus, for instance, a man of twenty-eight or twenty-nine may have arteries of sixty, and a man of forty may present vessels as much degenerated as they should be at eighty. Entire families sometimes show this tendency to early arterio-sclerosis, a tendency which cannot be explained in any other way than that in the make-up of the machine bad material was used for the tubing.

More commonly the arterio-sclerosis results from the bad use of good vessels, and among the circumstances which tend to produce this condition are the following:

(2) *Chronic Intoxications.*—Alcohol, lead, gout, and syphilis play an important *rôle* in the causation of arterio-sclerosis, although the precise mode of their action is not yet very clear. They may act, as Traube suggests, by increasing the peripheral resistance in the smaller vessels and in this way raising the blood tension, or possibly, as Bright taught, they alter

the quality of the blood and render more difficult its passage through the capillaries.

The poison of syphilis and of gout may act directly on the arteries, producing degenerative changes in the media and adventitia.

(3) *Overeating.*—Many authors attribute an important part of the etiology of arterio-sclerosis to the overfilling of the blood-vessels which occurs when unnecessarily large quantities of food and drink are taken. Particularly is this the case in stout persons who take very little exercise.

(4) *Overwork of the muscles*, which acts by increasing the peripheral resistance and by raising the blood-pressure.

(5) *Renal Disease.*—The relation between the arterial and kidney lesions has been much discussed, some regarding the arterial degeneration as secondary, others as primary. There are certainly two groups of cases, one in which the arterio-sclerosis is the first change, and the other in which it appears to be secondary to a primary affection of the kidneys. The former occurs, I believe, with much greater frequency than has been supposed.

Morbid Anatomy.—Thoma divides the cases into *primary* arterio-sclerosis, in which there are local changes in the arteries leading to dilatation and a compensatory increase of the connective tissue of the intima; *secondary* arterio-sclerosis, due to changes in the arteries which follow increased resistance to the blood-flow in the peripheral vessels. This increased tension leads to dilatation and to slowing of the blood-stream and a secondary compensatory development of the intima.

In a recent study of 41 autopsies upon arterio-sclerotic cases from my wards, Councilman * follows the useful division into nodular, senile, and diffuse forms.

(*a*) *Nodular Form.*—In the circumscribed or nodular variety the macroscopic changes are very characteristic. The aorta presents, in the early stages, from the ring to bifurcation, numerous flat projections, yellowish or yellowish white in color, hemispherical in outline, and situated particularly about the orifices of the branches. In the early stage these patches are scattered and do not involve the entire intima. In more advanced grades the patches undergo atheromatous changes. The material constituting the button undergoes softening and breaks up into granular material, consisting of molecular *débris*—the so-called atheromatous abscess.

In the circumscribed or nodular arterio-sclerosis the primary alteration consists in a degeneration or a local infiltration in the media and adventitia, chiefly about the vasa vasorum. The affection is really a mesarteritis and a periarteritis. These changes lead to the weakening of the wall in the affected area, at which spot the proliferative changes commence in the intima, particularly in the subendothelial structures, with gradual thick-

* Transactions of the Association of American Physicians, vol. vi.

ening and the formation of an atheromatous button or a patch of nodular arterio-sclerosis. The researches of Thoma have shown that this is really a compensatory process, and that before its degeneration the nodular button, which post mortem projects beyond the lumen, during life fills up and obliterates what would otherwise be a depression of the wall in consequence of the weakening of the media. A similar process goes on in the smaller vessels, and in any one of the smaller branches it can be readily seen on section that each patch of endarteritis corresponds to a defect in the media and often to changes in the adventitia. The condition is one which may lead to rapid dilatation or to the production of an aneurism, particularly in the early stage, before the weakened spot is thickened and strengthened by the intimal changes.

(*b*) *Senile Arterio-sclerosis.*—The larger arteries are dilated and tortuous, the walls thin but stiff, and often converted into rigid tubes. The subendothelial tissue undergoes degeneration and in spots breaks down, forming the so-called atheromatous abscesses, the contents of which consist of a molecular *débris*. They may open into the lumen, when they are known as atheromatous ulcers. The greater portion of the intima may be occupied by rough calcareous plates, with here and there fissures and losses of substance, upon which not infrequently white thrombi are deposited. Microscopically there is extreme degeneration of the coats, particularly of the media. Senile atrophy of the liver and kidneys usually accompanies these changes. Senile changes are common in other organs. The heart may be small and is not necessarily hypertrophied. In 7 of 14 cases of Councilman's series there was no enlargement. Brown atrophy is common

(*c*) *Diffuse Arterio-sclerosis.*—The process is wide-spread throughout the aorta and its branches, in the former usually, but not necessarily, associated with the nodular form. The subjects of this variety are usually middle-aged men, but it may occur early. Of the 27 in Councilman's series belonging to this group the majority were between the ages of forty and fifty-five. The youngest was a negro of twenty-three and the oldest a man of sixty. The affection is very prevalent among negroes; less than fifty per cent were in whites, whereas the ratio of colored to white patients in the wards is one to seven. The affection is met with in strongly built, muscular men and, as Councilman remarks, they rarely present on the autopsy table signs of general anasarca or, if œdema exists, it has come on during the last few days of life.

The aorta and its branches are more or less dilated, the branches sometimes more than the trunk. The intima may be smooth and show very slight changes to the naked eye; more commonly there are scattered elevated areas of an opaque white color, some of which may have undergone atheromatous changes as in the senile form. Microscopically the *media* shows necrotic and hyaline changes, involving in the larger arteries both muscular and elastic elements, and the *intima* presents a great increase

in the subendothelial connective tissue, which is particularly marked opposite areas of advanced degeneration in the media. The small arteries—those of the kidneys, for example—show "a thickening of the wall, due to the formation of a homogeneous hyaline tissue within the muscular coat. This tissue contains but few cells, is faintly striated, and stains a light brown in the osmic acid used in the hardening solution. In many of the smallest vessels nothing can be seen of the elastic lamina, in others only fragments can be made out, in others it is preserved. . . . The muscular fibres of the media show marked atrophic changes. Fatty degeneration of the cells can be made out both in fresh sections and after hardening in Fleming's solution. The nuclei are thin and atrophic and vacuoles are sometimes seen in them. In some arteries the muscle-fibres have almost disappeared and the media is changed into a homogeneous tissue, similar to that in the thickened intima" (Councilman). The degeneration of the media is most marked in the smaller arteries. The capillaries are thickened, particularly those of the glomeruli of the kidneys, which are often obliterated and involved in extensive hyaline degeneration.

It is in this group of cases that the heart shows the most important changes. The average weight in the cases referred to was over 450 grammes, and there were two cases in which without valvular disease the weight was over 800 grammes. Fibrous myocarditis is often present, particularly when the coronary arteries are involved. The semilunar valves are sometimes opaque and sclerotic, and may be incompetent. The kidneys may show extensive sclerosis, but in many cases the changes are so slight that macroscopically they might be overlooked. They may be increased in size. The capsule is usually adherent, the surface a little rough, and very often presents atrophic areas at a lower level of a deep-red color. Increased consistence is always present.

Sclerosis of the pulmonary artery is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral-valve disease and in emphysema. Sometimes the sclerosis reaches a high grade and is accompanied with aneurismal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve. In a remarkable case of a young man of twenty-four, reported by Romberg from Curschmann's clinic, the pulmonary arteries were involved in most extensive arterio-sclerosis; the main branches were dilated, and the smaller branches were the seat of the most extreme sclerotic changes. On the other hand, the aorta and its branches were normal. The heart was greatly hypertrophied, and the clinical symptoms were those of a congenital heart affection. In many cases of arterio-sclerosis the condition is not confined to the arteries, but extends not only to the capillaries but also to the veins, and may properly be termed *angio-sclerosis.*

Sclerosis of the veins—*phlebo-sclerosis*—is not at all an uncommon accompaniment of arterio-sclerosis, and is a condition to which of late a good deal of attention has been paid. It is seen in conditions of height-

ened blood-pressure, as in the portal system in cirrhosis of the liver and in the pulmonary veins in mitral stenosis. The affected vessels are usually dilated, and the intima shows, as in the arteries, a compensatory thickening, which is particularly marked in those regions in which the media is thinned. The new-formed tissue in the endophlebitis may undergo hyaline degeneration, and is sometimes extensively calcified. In a case of fibroid obliteration of the portal vein of long standing, I found the intima of the greatly dilated gastric, splenic, and mesenteric extensively calcified. In ordinary diffuse arterio-sclerosis the veins may also be involved, but rarely to a marked degree.

Symptoms.—Many patients never come under observation during life, but are seen for the first time on the post-mortem table, having died suddenly from cerebral hæmorrhage, blocking of a coronary artery, or rupture of an aneurism.

Among important symptoms are the following:

Increased Tension.—The pressure with which the blood flows in the arteries depends upon the degree of peripheral resistance and the force of the ventricular contraction. A high-tension pulse may exist with very little arterio-sclerosis; but, as a rule, when the condition has been persistent, the sclerosis and high tension are found together. The pulse wave is slow in its ascent, enduring, subsides slowly, and in the intervals of the beats the vessel remains full and firm. It may be very difficult to obliterate the pulse, and the firmest pressure on the radial or the temporal may not be sufficient to annihilate the pulse wave beyond the point of pressure. The sphygmographic tracing shows a sloping, short upstroke, no percussion wave, and a slow, gradual descent, in which the dicrotic wave is very slightly marked. It may be difficult to estimate how much of the hardness and firmness is due to the tension of the blood within the vessel, and how much to the thickening of the wall. If, for example, when the radial is compressed with the index-finger the artery can be felt beyond the point of compression, its walls are sclerosed.

Hypertrophy of the Heart.—In consequence of the peripheral resistance and increased work the left ventricle increases in size, and some of the purest examples of simple hypertrophy occur in this condition. The chamber may be little, if at all, dilated. The apex beat is dislocated in advanced cases an inch or more beyond the nipple line. The impulse is heaving and forcible. The aortic second sound is clear, ringing, and accentuated.

The combination of increased arterial tension, a palpable thickening of the arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are signs pathognomonic of arterio-sclerosis. From this period of establishment the course of the disease may be very varied. For years the patient may maintain good health, and be in a condition analogous to a person with a well-compensated valvular lesion. There may be no renal symptoms, or there may be the passage of a larger

^ Occasionally.

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Published in Lancet, 1912, ii, p. 1463.

Cerebral angiospasm

To the Editor of the Lancet.

Sir

As Sir William Russell, having had the good fortune to graduate from Edinburgh University, before any of the text-book which did not appear until 1892, but having written so much and so well on cerebro-sclerosis that he might have read the section on the subject, before saying (Lancet, Nov. 16th) in some one of the eight editions,

"How little the reference communication of Sir Peabody influenced medical teaching or thinking is shown by the absence of any reference to this condition in Sir William Osler's account of cerebral [illegible] in the 1909 edition, at least so far as I have been able to discover".

Any knowledge of transient aphasia

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amount of urine than normal, with transient albuminuria, and now and then hyaline tube-casts. The subsequent history is extraordinarily diverse, depending upon the vascular territory in which the sclerosis is most advanced, or upon the accidents which are so liable to happen, and the symptoms may be cardiac, cerebral, renal, etc.

(1) *Cardiac.*—The involvement of the coronary arteries may lead to the various symptoms already referred to under that section—thrombosis with sudden death, fibroid degeneration of the heart, aneurism of the heart, rupture, and angina pectoris. Angina pectoris is extremely common, and in the true variety is almost always associated with arterio-sclerosis. A second important group of cardiac symptoms results from the dilatation which ultimately may follow the hypertrophy. The patient then presents all the symptoms of cardiac insufficiency—dyspnœa, scanty urine, and very often serous effusions. If the case has come under observation for the first time the clinical picture is that of chronic valvular disease, and the existence of a loud blowing murmur at the apex may throw the practitioner off his guard. Many cases terminate in this way.

(2) The *cerebral* symptoms of arterio-sclerosis are varied and important, and embrace those of many degenerative processes, acute and chronic (which follow sclerosis of the smaller branches), and cerebral hæmorrhage, which is usually associated with the miliary aneurisms.

Transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. Recovery may be perfect. It is difficult to say upon what these attacks depend. Spasm of the arteries has been suggested, but the condition of the smaller arteries is not very favorable to this view. Peabody has recently called attention to these cases, which are more common than indicated in the literature.

(3) *Renal* symptoms supervene in a large number of the cases. A sclerosis, patchy or diffuse, is present in a majority of the cases at the time of autopsy, and the condition is practically that of contracted kidneys. It is seen in a typical manner in the senile form, and not infrequently develops early in life as a direct sequence of the diffuse variety. It is often difficult to decide clinically (and the question is one upon which good observers might not agree in a given case) whether the arterial or the renal disease has been primary.

(4) Among other events in arterio-sclerosis may be mentioned gangrene of the extremities, due either directly to endarteritis or to the dislodgment of thrombi. Respiratory symptoms are not uncommon, particularly bronchitis and the symptoms associated with emphysema.

Treatment.—In the late stages the conditions must be treated as they arise in connection with the various viscera. In the early stages, before any local symptoms are manifest, the patient should be enjoined to live a quiet, well-regulated life, avoiding excesses in food and drink. It is usually best to explain frankly the condition of affairs, and so gain his intelligent co-operation. Special attention should be paid to the state of

the bowels and urine, and the secretion of the skin should be kept active by daily baths. Alcohol in all forms should be prohibited, and the food should be restricted to plain, wholesome articles. The use of mineral waters or a residence every year at one of the mineral springs is usually serviceable. If there has been a syphilitic history an occasional course of iodide of potassium is indicated, and whenever the pulse tension is high nitroglycerine may be used.

In cases which come under observation for the first time with dyspnœa, slight lividity, and signs of cardiac insufficiency, venesection is indicated. In some instances, with very high tension, striking relief is afforded by the abstraction of twenty ounces of blood.

III. ANEURISM.

The following forms of aneurism are usually recognized:

(*a*) The *true*, in which the sac is formed of one or more of the arterial coats. This may be fusiform, cylindrical, or cirsoid (in which the dilatation is in an artery and its branches), or it may be circumscribed or sacculated. Aneurisms are usually fusiform, resulting from uniform dilatation of the vessel, or saccular.

(*b*) The *false* or *dissecting* aneurism, which results from injury or laceration of the internal coat. The blood dissects between the layers; hence the name, dissecting aneurism. This occurs usually in the aorta. It may dissect the entire length of the vessel, and, perforating into the lumen of the vessel, may, as in a case reported by J. E. Graham, persist for years.

(*c*) *Arterio-venous* aneurism results when a communication is established between an artery and a vein. A sac may intervene, in which case it is called a varicose aneurism; but in many cases the communication is direct and the chief change is in the vein, which is dilated, tortuous, and pulsating, and is termed an aneurismal varix.

Etiology and Pathology.—Aneurisms arise: (*a*) By the gradual diffuse distention of the arterial coats, which have been weakened by arterio-sclerosis, particularly in its early stages, before compensatory endarteritis develops. The arch of the aorta is often dilated in this way so as to form an irregular aneurism.

(*b*) In consequence of circumscribed loss of resisting power in the media and adventitia, and due often to laceration of the media. This is the most common cause of sacculated aneurism. The laceration is frequently found in the ascending portion of the arch and occurs early in the process of arterio-sclerosis, before the compensatory thickening has taken place. Occasionally one meets with remarkable specimens illustrating the important part played by this process. The intima may also be torn. In a case of Daland's there was just above the aortic valves

Should not the false aneurism be the one with scar tissue?

Traction aneurism of the aorta. Thoma, Virchow's Archiv, Bd. 122. In his case the lesion was in the body of a young man, aged 26, dead of typhoid fever, who had an old kypho-scoliosis with displacement of the thoracic viscera. The aneurism was at the concavity of the aortic arch at the point of the insertion of the ligament representing the remnants of the ductus Botalli. Thoma thought that in the kypho-scoliosis and displacement of the viscera the pulmonary artery tended to shorten the ligament of Botalli, and as the aorta was relatively fixed to the vertebral column by the intercostal arteries it suffered traction.

an old transverse tear of the intima, extending almost the entire circumference of the vessel. Sclerosis of the media and adventitia had taken place and the process was evidently of some standing. An inch or more above it was a fresh transverse rent which had produced a dissecting aneurism. These arterio-sclerotic aneurisms, as they are called, are found also in the smaller vessels.

(*c*) *Embolic Aneurism.*—When an embolus has lodged in a vessel and permanently plugged it, aneurismal dilatation may follow on the proximal side. The embolus itself may, if a calcified fragment from a valve, lacerate the wall, or if infected may produce inflammation and softening. In either case aneurism may result.

(*d*) *Mycotic Aneurism.*—The importance of this form has been specially considered by Eppinger in his exhaustive monograph. The occurrence of multiple aneurisms in malignant endocarditis has been observed by several writers. Probably the first case in which the mycotic nature was recognized was one which occurred at the Montreal General Hospital and is reported in full in my lectures on malignant endocarditis. In addition to the ulceration of the valves there were four aneurisms of the arch, of which one was large and saccular, and three were not bigger than cherries. An extensive growth of micrococci was present in the larger as well as in the smaller sacs.

A form of parasitic aneurism which occurs with great frequency in the mesenteric arteries of the horse is due to the development of the *strongylus armatus*.

And, lastly, there are cases in which without any definite cause there is a tendency to the development of aneurisms in various parts of the body. A remarkable instance of it in our profession was afforded by the brilliant Thomas King Chambers, who first had an aneurism in the left popliteal artery, eleven years subsequently an aneurism in the right leg which was cured by pressure, and finally aneurism of both carotid arteries.

Aneurism of the Thoracic Aorta.

The causes which favor the development of arterio-sclerosis prevail in aortic aneurism, particularly alcohol, syphilis, and overwork. The greatest danger probably is in strong muscular men with commencing degenerative processes in the arteries (a consequence of syphilis or alcohol or a result of hereditary weakness of the arterial tissues), who during a sudden muscular exertion are liable to lacerate the media, the intima not yet being strengthened by compensatory thickening over a spot of mesarteritis. Aneurisms of the thoracic aorta vary greatly in size and shape. A majority of them are saccular. They may be small and situated just above the aortic ring. Others form large tumors which project externally and occupy a large portion of the upper thorax. Small sacs from the descending portion of the arch may compress the trachea or the bronchi. In the tho-

racic portion the sac may erode the vertebræ or grow into the pleural cavity and compress the lung. In some instances it grows through the ribs and appears in the back.

Symptoms.—The chief influence of an aneurism is manifested in what are known as pressure effects. In the absence of these the aneurisms attain a large size without producing symptoms or seriously interfering with the circulation. Indeed, a useful clinical subdivision as given by Bramwell is into three groups—aneurisms which are entirely latent and give no physical signs; aneurisms which present signs of intrathoracic pressure, but it is difficult or impossible to determine the nature of the lesion producing the pressure; and, lastly, aneurisms which produce distinct tumors with well-marked pressure symptoms and external signs. It is perhaps best to consider aneurisms of the aorta according to the situation of the tumor.

(*a*) *Aneurisms of the Ascending Portion of the Arch.*—When just above the sinuses of Valsalva they are often small and latent. The first symptom may be rupture, which usually takes place into the pericardium and causes instant death. Above the sinuses, along the convex border of the ascending part, aneurism frequently develops, and may grow to a large size, either passing out into the right pleura or forward, pointing at the second or third interspace, eroding the ribs and sternum, and producing large external tumors. In this situation the sac is liable indeed to compress the superior vena cava, causing engorgement of the vessels of the head and arm, sometimes compressing only the subclavian vein, and causing enlargement and œdema of the right arm. Perforation may take place into the superior vena cava, of which accident Pepper and Griffith have collected twenty-nine cases. Large aneurisms in this situation may cause much dislocation of the heart, pushing it down and to the left, and sometimes compressing the inferior vena cava, and causing swelling of the feet and ascites. The right recurrent laryngeal nerve is often involved in these tumors. Death commonly follows from rupture into the pleura, or into the superior cava; less commonly from rupture externally, sometimes from heart-failure.

(*b*) *Aneurisms of the Transverse Arch.*—These may grow forward, erode the sternum, and produce large tumors. More commonly they are small and produce no external tumor, but cause marked pressure signs in their growth backward toward the spine, involving the trachea and the œsophagus, producing cough, which is often of a paroxysmal character, and dysphagia. The left recurrent laryngeal is often involved in its course round the arch. A small aneurism from the lower or posterior wall of the arch may compress a bronchus, inducing bronchorrhœa, gradual bronchiectasy, and suppuration in the lung—a process which by no means infrequently causes death in aneurism, and a condition which at the Montreal General Hospital we were in the habit of terming aneurismal phthisis. Occasionally enormous aneurisms develop in this situa-

tion, and grow into both pleuræ, extending between the manubrium and the vertebræ, and may persist for years. The sac may be evident at the sternal notch. The innominate, less commonly the left carotid and subclavian, may be involved in the sac, and the radial or carotid pulse may be absent or retarded. Pressure on the sympathetic may at first cause dilatation and subsequently contraction of the pupil. Sometimes the thoracic duct is compressed.

(*c*) *Aneurisms of the Descending Portion.*—Pressure signs are not so marked. The pain is often intense, owing to erosion of the vertebræ. Dysphagia may occur. Compression of the lung or compression of certain bronchi may induce bronchiectasy, retention of secretions, and fever. A tumor may appear externally in the region of the scapula, and here attain an enormous size. Occasionally the aneurisms in this region are small and latent, and prove fatal by rupture into the œsophagus. I have reported a case of sudden death, in which the heart and arch of the aorta were normal and the stomach was distended with blood, which could not be accounted for until the œsophagus was slit open, when it was found that a small aneurism in the thoracic aorta, smaller than a walnut, had ruptured into the gullet. The sac may erode the vertebræ and open the spinal canal, producing compression of the cord. Death not infrequently occurs from rupture into the pleura.

Diagnosis and Physical Signs.—*Inspection.*—In many instances this is negative. On either side of the sternum there may be abnormal pulsation, due to dislocation of the heart or to deformity of the thorax. The aneurismal pulsation is usually above the level of the third rib and most commonly to the right of the sternum, either in the first or second interspace. It may be only a diffuse heaving impulse without any external tumor. Often the impulse is noticed only when the chest is looked at obliquely in a favorable light. When the innominate is involved the throbbing may pass into the neck or be apparent at the sternal notch. Posteriorly, when pulsation occurs, it is most commonly found in the left scapular region. An external tumor is present in many cases, projecting either through the upper part of the sternum or to the right, sometimes involving the sternum and costal cartilages on both sides, forming a tumor the size of a cocoa-nut or even larger. The skin is thin, often blood-stained, or it may have ruptured, exposing the laminæ of the sac. The apex beat may be much dislocated, particularly when the sac is large. It is more commonly a dislocation from pressure than from enlargement of the heart itself.

Palpation.—The area and degree of pulsation are best determined by palpation. When the aneurism is deep-seated and not apparent externally, the bimanual method should be used, one hand upon the spine and the other on the sternum. When the sac has perforated the chest wall the impulse is, as a rule, forcible, slow, heaving, and expansile. The resistance may be very great if there are thick laminæ beneath the skin;

more rarely the sac is soft and fluctuating. The hand upon the sac, or on the region in which it is in contact with the chest wall, feels in many cases a diastolic shock, often of great intensity, which forms one of the valuable physical signs of aneurism. A systolic thrill is sometimes present, not so often in saccular aneurisms as in the dilatation of the arch. The pulsation may sometimes be felt in the suprasternal notch.

Percussion.—The small and deep-seated aneurisms are in this respect negative. In the larger tumors, as soon as the sac reaches the chest wall, there is produced an area of abnormal dulness, the position of which depends upon the part of the aorta affected. Aneurisms of the ascending arch grow forward and to the right, producing dulness on one side of the manubrium; those from the transverse arch produce dulness in the middle line, extending toward the left of the sternum, while aneurisms of the descending portion most commonly produce dulness in the left interscapular and scapular regions. The percussion note is flat and gives a feeling of increased resistance.

Auscultation.—Adventitious sounds are not always to be heard. Even in a large sac there may be no murmur. Much depends upon the thickness of the laminæ of fibrin. An important sign, particularly if heard over a dull region, is a ringing, accentuated second sound, a phenomenon rarely missed in large aneurisms of the aortic arch. A systolic murmur may be present; sometimes a double murmur, in which case the diastolic *bruit* is usually due to associated aortic insufficiency. The systolic murmur alone is of little moment in the diagnosis of an aneurismal sac. With the single stethoscope the shock of the impulse with the first sound is sometimes very marked.

Among other physical signs of importance are slowing of the pulse in the arteries beyond the aneurism, or in those involved in the sac. There may, for instance, be a marked difference between the right and left radial, both in volume and time. A physical sign of large thoracic aneurism which I have not seen referred to is obliteration of the pulse in the abdominal aorta and its branches. My attention was called to this in a patient who was stated to have aortic insufficiency. There was a well-marked diastolic murmur, but in the femorals and in the aorta I was surprised to find no trace of pulsation, and not the slightest throbbing in the abdominal aorta or in the peripheral arteries of the leg. The circulation was, however, unimpaired in them and there was no dilatation of the veins. Attracted by this, I then made a careful examination of the patient's back, when the circumstance was discovered, which neither the patient himself nor any of his physicians had noticed, that he had a very large area of pulsation in the left scapular region. The sac probably was large enough to act as a reservoir annihilating the ventricular systole, and converting the intermittent into a continuous stream.

The *tracheal tugging*, a valuable sign in deep-seated aneurisms, was described by Surgeon-Major Porter, and has been specially studied by my

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colleagues Ross and MacDonnell * at the Montreal General Hospital. To test it the patient should sit up with the head inclined forward, so as completely to relax the neck. The cricoid cartilage is grasped between the index-finger and the thumb and by upward pressure the trachea put upon the stretch. In healthy individuals no sensation is felt, but if an aneurism is attached to it or is adherent in the immediate vicinity, the stretching is accompanied by a well-marked and characteristic tugging. On several occasions I have known this to be a sign of great value in the diagnosis of deep-seated aneurisms. I have never felt it in tumors, or in the extreme dynamic dilatation of aortic insufficiency.

Occasionally a systolic murmur may be heard in the trachea, as pointed out by David Drummond, or even at the patient's mouth, when opened. This is either the sound conveyed from the sac, or is produced by the air as it is driven out of the wind-pipe during the systole.

An important but variable feature in thoracic aneurism is *pain*, which is particularly marked in deep-seated tumors. It is usually paroxysmal, sharp, and lancinating, often very severe when the tumor is eroding the vertebræ, or perforating the chest wall. In the latter case, after perforation the pain may cease. Anginal attacks are not uncommon, particularly in aneurisms at the root of the aorta. Frequently the pain radiates down the left arm or up the neck, sometimes along the upper intercostal nerves. *Cough* results either from the direct pressure on the wind-pipe, or is associated with bronchitis. The expectoration in these instances is abundant, thin, and watery; subsequently it becomes thick and turbid. Paroxysmal cough of a peculiar brazen, ringing character is a characteristic symptom in some cases, particularly when there is pressure on the recurrent laryngeal nerves.

Dyspnœa, which is common in cases of aneurism of the transverse portion, is not necessarily associated with pressure on the recurrent laryngeal nerves, but may be due directly to compression of the trachea or the left bronchus. It may occur with marked stridor. Loss of voice and hoarseness are consequences of pressure on the recurrent laryngeal, usually the left, inducing either a spasm in the muscles of the left vocal cord or paralysis.

Paralysis of an abductor on one side may be present without any symptoms. It is more particularly, as Semon states, when the paralytic contractures supervene that the attention is called to laryngeal symptoms.

Hæmorrhage in thoracic aneurism may come from (*a*) the soft granulations in the trachea at the point of compression, in which case the sputa are blood-tinged, but large quantities of blood are not lost; (*b*) from rupture of the sac into the trachea or bronchi; (*c*) from perforation into the lung or erosion of the lung tissue. The bleeding may be profuse, rapidly proving fatal, and is a common cause of death. It may persist for weeks or

* London Lancet, 1891.

months, in which case it is simply hæmorrhagic weeping through the sac, which is exposed in the trachea. In some instances, even after a very profuse hæmorrhage, the patient recovers and may live for years. A patient with well-marked thoracic aneurism, whom I showed to my class at the University of Pennsylvania four years ago and who had had several brisk hæmorrhages, died recently, having in the mean time enjoyed average health.

Difficulty of swallowing is a comparatively rare symptom, and may be due either to spasm or to direct compression. The sound should never be passed in these cases, as the œsophagus may be almost eroded and a perforation may be made.

Among other signs and symptoms venous compression, which has already been mentioned, may involve one subclavian or the superior vena cava. A curious phenomenon in intrathoracic aneurism is the clubbing of the fingers and incurving of the nails of one hand, of which two examples have been under my care, in both without any special distention or signs of venous engorgement. Tumors of the arch may involve the pulmonary artery, producing compression, or in some instances adhesion of the pulmonary segments and insufficiency of the valve; or the sac may rupture into the artery, an accident which happened in two of my cases, producing instantaneous death.

Pressure on the sympathetic is particularly liable to occur in growths from the ascending portion of the arch. Either the upper dorsal or the lower cervical ganglion is involved. The symptoms are variable. If the nerve is simply irritated there is stimulation of the vaso-dilator fibres and dilatation of the pupil. With this may be associated pallor of the same side of the face. On the other hand, destruction of the cilio-spinal branches causes paralysis of the dilator fibres, in consequence of which the iris contracts, the vessels on the side of the head dilate, causing congestion, and in some instances unilateral sweating. It is much more common to see the pupillary symptoms alone than in combination either with pallor, redness, or sweating.

The clinical picture of aneurism of the aorta is extremely varied. Many cases present characteristic symptoms and no physical signs, while others have well-marked physical signs and no symptoms. As Broadbent remarks, the aneurism of *physical signs* springs from the ascending portion of the aorta; the aneurism of *symptoms* grows from the transverse arch.

Aneurism of the aorta may be confounded with: (*a*) The violent throbbing impulse of the arch in aortic insufficiency. I have already referred to a case of this kind in which the diagnosis of aneurism was made by several good observers. In a case recently under observation dulness and pulsation existed in the second right interspace with a well-marked systolic and a loud diastolic murmur, which was heard far out in the right mammary region. The question arose whether aneurism was present in

Machine: Cobla

addition to the aortic insufficiency. The post-mortem showed the margin of the right lung retracted and adherent to the pericardium, leaving exposed the aorta, which must have been greatly distended during each systole.

(*b*) *Simple Dynamic Pulsation.*—No instance of this, which is common in the abdominal aorta, has ever come under my notice. One which came under the care of William Murray and Bramwell presented, without any pain or pressure symptoms, pulsation and dulness over the aorta. The condition gradually disappeared and was thought to be neurotic.

(*c*) Dislocation of the heart in curvature of the spine may cause great displacement of the aorta, so that it has been known to pulsate forcibly to the right of the sternum.

(*d*) *Solid Tumors.*—When the tumor projects externally and pulsates the difficulty may be considerable. In tumor the heaving, *expansile* pulsation is absent, and there is not that sense of force and power which is so striking in the throbbing of a perforated aneurism. There is not to be felt as in aortic aneurism the shock of the heart-sounds, particularly the diastolic shock. Auscultatory sounds are less definite, as large aneurisms may occur without murmur; and, on the other hand, murmurs may be heard over tumors. The greatest difficulty is in the deep-seated thoracic tumors, and here the diagnosis may be impossible. I have already referred to the case which was regarded by Skoda as aneurism and by Oppolzer as tumor. The physical signs may be indefinite. The ringing aortic second sound is of great importance and is rarely, if ever, heard over tumor. Tracheal tugging is here a valuable sign. Pressure phenomena are less common in tumor, whereas pain is more frequent. The general appearance of the patient in aneurism is much better than in tumor. There may be signs of enlargement of the glands in the axilla or in the neck. Healthy, strong males who have worked hard and have had syphilis are the most common subjects of aneurism. Occasionally cancer of the œsophagus may simulate aneurism, producing pressure on the left bronchus, and in one instance at the Philadelphia Hospital, with a husky, brazen cough, the symptoms were very suggestive.

(*e*) *Pulsating Pleurisy.*—In cases of *empyema necessitatis*, if the projecting tumor is in the neighborhood of the heart and pulsates, the condition may readily be mistaken for aneurism. The absence of the heaving, firm distention and of the diastolic shock would, together with the history and the existence of pleural effusion, determine the nature of the case. If necessary, puncture may be made with a fine hypodermic needle. In a majority of the cases of pulsating pleurisy the throbbing is diffuse and widespread, moving the whole side.

Prognosis.—The outlook in thoracic aneurism is always grave. Life may be prolonged for some years, but the patients are in constant jeopardy. Spontaneous cure is not very infrequent in the small sacculated tumors of the ascending and thoracic portions. The cavity becomes filled with lam-

inæ of firm fibrin, which become more and more dense and hard, the sac shrinks considerably, and finally lime salts are deposited in the old fibrin. The laminæ of fibrin may be on a level with the lumen of the vessel, causing complete obliteration of the sac. The cases which rupture externally, as a rule run a rapid course, although to this there are exceptions; the sac may contract, become firm and hard, and the patient may live for five, or even, as in a case mentioned by Balfour, for ten years. The cases which have lasted longest in my experience have been those in which a saccular aneurism has projected from the ascending arch. One patient in Montreal had been known to have aneurism for eleven years. The aneurism may be enormous, occupying a large area of the chest, and yet life be prolonged for many years, as in the case mentioned as under the care of Skoda and Oppolzer. One of the most remarkable instances is the case of dissecting aneurism reported by Graham. The patient was invalided after the Crimean War with aneurism of the aorta, and for years was under the observation of J. H. Richardson, of Toronto, under whose care he died in 1885. The autopsy showed a healed aneurism of the arch, with a dissecting aneurism passing the whole length of the aorta, which formed a double tube.

Treatment.—In a large proportion of the cases this can only be palliative. Still in every case measures should be taken which are known to promote clotting and consolidation within the sac. In any large series of cured aneurisms a considerable majority of the patients have not been known to be subjects of the disease, but the obliterated sac has been found accidentally at the post mortem.

The most satisfactory plan in early cases, when it can be carried out thoroughly, is that advised by the late Mr. Tufnell, of Dublin, the essentials of which are rest and a restricted diet. Rest is essential and should, as far as possible, be absolute. The reduction of the daily number of heart-beats when a patient is recumbent and makes no exertion whatever amounts to many thousands, and is one of the principal advantages of this plan. Mental quiet should also be enjoined. The diet advised by Tufnell is extremely rigid—for breakfast, two ounces of bread and butter and two ounces of milk; for dinner, two or three ounces of meat and three or four ounces of milk or claret; for supper, two ounces of bread and two ounces of milk. This low diet diminishes the blood-volume and is thought also to render the blood more fibrinous. It reduces greatly the blood-pressure within the sac, in this manner favoring coagulation. This treatment should be pursued for several months, but, except in persons of a good deal of mental stamina, it is impossible to carry it out for more than a few weeks at a time. It is a form of treatment adapted only for the saccular form of aneurism, and in cases of large sacs communicating with the aorta by a comparatively small orifice the chances of consolidation are fairly good. Unquestionably rest and the restriction of the liquids are the important parts of the treatment, and a greater

variety and quantity of food may be allowed with advantage. If this plan cannot be thoroughly carried out, the patient should at any rate be advised to live a very quiet life, moving about with deliberation and avoiding all sudden mental or bodily excitement. The bowels should be kept regular, and constipation and straining should be carefully avoided. Of medicines, iodide of potassium, as advised by Balfour, is of great value. It may be given in doses of from ten to fifteen or twenty grains three times a day. Larger doses are not necessary. The mode of action is not well understood. It may act by increasing the secretions and so inspissating the blood, by lowering the blood-pressure, or, as Balfour thinks, by causing thickening and contraction of the sac. The most striking effect of the iodide in my experience has been the relief of the pain. The evidence is not conclusive that the syphilitic cases are more benefited than the non-syphilitic. All these measures have little value unless the sac is of a suitable form and size. The large tumors with wide mouths communicating with the ascending portion of the aorta may be treated on the most approved plans for months without the slightest influence other than reduction in the intensity of the throbbing. A patient with a tumor projecting into the right pleura remained on the most rigid Tufnell treatment for more than one hundred days, during which time he also took iodide of potassium faithfully. The pulsations were greatly reduced and the area of dulness diminished, and we congratulated ourselves that the sac was probably consolidating. Sudden death followed rupture into the pleura, and the sac contained only fluid blood, not a shred of fibrin. In cases in which the tumor is large, or in which there seems to be very little prospect of consolidation, it is perhaps better to advise a man to go on quietly with his occupation, avoiding excitement and worry. Our profession has offered many examples of good work thoroughly and conscientiously carried out by men with aneurism of the aorta, who wisely, I think, as the late Hilton Fagge, preferred to die in harness. Other measures to induce coagulation in the sac are electricity, which has occasionally proved successful; the insertion of horse-hair, thin wire, or needles; the injection of an astringent liquid, such as perchloride of iron, into the sac. In a few cases only these have been followed by cure. The fine silver wire pushed through a hypodermic needle is probably the most satisfactory method, and may be combined with electrolysis, the method known as Loreta's. Kerr and Rosenstein, of San Francisco, have recently reported cases in which cure was effected in this way.

Other Symptoms requiring Treatment.—Pressure on veins causing engorgement, particularly of the head and arms, is sometimes promptly relieved by free venesection, and at any time during the course of a thoracic aneurism, if attacks of dyspnœa with lividity supervene, bleeding may be resorted to with great benefit. It has the advantage also of promptly checking the pain, for which symptom, as already mentioned, the iodide of potassium often gives relief. In the final stages morphia is, as a

rule, necessary. Dyspnœa, if associated with cyanosis, is best relieved by bleeding. Chloroform inhalations may be necessary. The question sometimes comes up with reference to tracheotomy in these cases of urgent dyspnœa. If it can be shown by laryngoscopic examination that it is due to bilateral abductor paralysis the trachea may be opened, but this is extremely rare, and in nearly every instance the urgent dyspnœa is caused by pressure about the bifurcation. When the sac appears externally and grows large an ice-cap may be applied upon it, or a belladonna plaster to allay the pain. In some instances an elastic support may be used with advantage, and I saw a physician with an enormous external aneurism in the right mammary region who for many months had obtained great relief by the elastic support, passing over the shoulder and under the arm of the opposite side.

Digitalis, ergot, aconite, and veratrum viride are rarely, if ever, of service in thoracic aneurism.

Aneurism of the Abdominal Aorta.

The sac is most common in the neighborhood of the cœliac axis. It is rare in comparison with thoracic aneurism. The tumor may be fusiform or sacculated, and it is sometimes multiple. Projecting backward, it erodes the vertebræ and may cause numbness and tingling in the legs and finally paraplegia, or it may pass into the thorax and burst into the pleura. More commonly the sac is on the anterior wall and projects forward as a definite tumor, which may be either in the middle line or a little to the left. The tumor may be large and evident, or when high up beneath the pillar of the diaphragm it may attain considerable size without being very apparent on palpation.

The symptoms are chiefly pain, very often of a cardialgic nature, passing round the sides or localized in the back, and gastric symptoms, particularly vomiting. Retardation of the pulse in the femoral is a very common symptom.

Diagnosis and Physical Signs.—Inspection may show marked pulsation in the epigastric region, sometimes a definite tumor. A thrill is not uncommon. The pulsation is forcible, expansile, and sometimes double when the sac is large and in contact with the pericardium. On palpation a *definite tumor can be felt.* If large, there is some degree of dulness on percussion which usually merges with that of the left lobe of the liver. On auscultation, a systolic murmur is, as a rule, audible, and is sometimes best heard at the back. A diastolic murmur is occasionally present, usually very soft in quality. One of the commonest of clinical errors is to mistake a throbbing aorta for an aneurism. It is to be remembered that no pulsation, however forcible, or the presence of a thrill or a systolic murmur justifies the diagnosis of abdominal aneurism unless there is a *definite tumor which can be grasped and which has an expansile pulsa-*

tion. Attention to this rule will save many errors. The throbbing or pulsating aorta is met with in all neurasthenic conditions, particularly in women, and it is remarkable with what violence the epigastrium may be driven out with each systole. In anæmia, particularly some instances of traumatic anæmia, the throbbing may be very great. In the case of a large, stout man with severe hæmorrhages from a duodenal ulcer the throbbing of the abdominal aorta not only shook violently the whole abdomen, but communicated a pulsation to the bed, the shock of which was distinctly perceptible to any one sitting upon it. Very frequently a tumor of the pylorus, of the pancreas, or of the left lobe of the liver is lifted with each impulse of the aorta and may be confounded with aneurism. The absence of the forcible expansile impulse and the examination in the knee-elbow position, in which the tumor, as a rule, falls forward, and the pulsation is not then communicated, suffice for differentiation.

The outlook in abdominal aneurism is bad. A few cases heal spontaneously. Death may result from (*a*) complete obliteration of the lumen by clots; (*b*) compression paraplegia; (*c*) rupture either into the pleura, retroperitoneal tissues, peritonæum or the intestines, very commonly the duodenum; (*d*) by embolism of the superior mesenteric artery, producing infarction of the intestines.

The *treatment* is such as already advised in thoracic aneurism. When the aneurism is low down pressure has been successfully applied in a case by Murray, of Newcastle. It must be kept up for many hours under chloroform. The plan is not without risk, as patients have died from bruising and injury of the sac.

Aneurism of the Branches of the Abdominal Aorta.

The *cœliac axis* is itself not infrequently involved in aneurism of the first portion of the abdominal aorta. Of its branches, the *splenic artery* is occasionally the seat of aneurism. This rarely causes tumor large enough to be felt; sometimes, however, the tumor is of large size. I have reported a case in a man, aged thirty, who had an illness of several months' duration, severe epigastric pain and vomiting, which led his physicians in New York to diagnose gastric ulcer. There was a deep-seated tumor in the left hypochondriac region, the dulness of which merged with that of the spleen. There was no pulsation, but it was thought on one occasion that a *bruit* was heard. The chief symptoms while under observation were vomiting, severe epigastric pain, occasional hæmatemesis, and finally severe hæmorrhage from the bowels. An aneurism of the splenic artery the size of a cocoa-nut was situated between the stomach above and the transverse colon below, and extended to the left as far as the level of the navel. The sac contained densely laminated fibrin. It had perforated the colon. I have twice seen small aneurisms on the splenic artery. Of thirty-nine instances

of aneurism on the branches of the abdominal aorta collected by Lebert, ten were of the splenic artery.

Aneurism of the *hepatic artery* is very rare, and there are only ten or twelve cases on record. The symptoms are extremely indefinite; the condition could rarely be diagnosed. In the case reported by Ross and myself, a man aged twenty-one had the symptoms of pyæmia. The liver was greatly enlarged, weighed nearly 5,000 grammes, and presented innumerable small abscesses. An oval aneurism, half the size of a small lemon, involved the right and part of the left branches.

A few cases of aneurism of the *superior mesenteric artery* are on record. The diagnosis is scarcely possible. Plugging of the branches or of the main stem may cause the symptoms of infarction of the bowels which have already been considered.

Small aneurisms of the *renal artery* are not very uncommon. Large tumors are rare. The sac may rupture and give rise to extensive retroperitoneal hæmorrhage.

Arterio-venous Aneurism.

In this form there is abnormal communication between an artery and a vein. When a tumor lies between the two it is known as varicose aneurism; when there is a direct communication without tumor the vein is chiefly distended and the condition is known as aneurismal varix.

An aneurism of the ascending portion of the arch may open directly into the vena cava. Twenty-nine cases of this lesion have been analyzed by Pepper and Griffith. Cyanosis, œdema, and great distention of the veins of the upper part of the body are the most frequent symptoms, and develop, as a rule, with suddenness. Of the physical signs a thrill is present in some cases. A continuous murmur with systolic intensification is of great diagnostic value. In a recent case, after the existence for some time of pressure symptoms, intense cyanosis developed with engorgement of the veins of the head and arms. Over the aortic region there was a loud continuous murmur with systolic intensification.

A majority of the cases of arterio-venous aneurism and of aneurismal varix result from the accidental opening of an artery and vein as in venesection, and are met with at the bend of the elbow or sometimes in the temporal region. The condition may persist for years without causing any trouble. Pulsation, a loud thrill, and a continuous humming murmur are usually present.

Congenital Aneurism.

In consequence of failure of proper development of the elastic coat in many places in the arterial system, multiple aneurisms may develop. In the well-known case described by Küssmaul and Maier, upon many of the

See admirable plate of varicose aneurism in Dorsey's Elements of Surgery.

medium-sized arteries there were nodular prominences, which consisted of thickening of the intima and infiltration of the adventitia and of the media, with a nuclear growth which in places looked quite sarcomatous. They called it a case of *periarteritis nodosa*, and Eppinger holds that it belongs to the category which he makes of congenital aneurism. As many as sixty-three aneurismal tumors have been found in one case. In the smaller branches, such as the coronary and the mesenteric arteries or in the pulmonary arteries, there may be numerous elongated or saccular aneurisms varying in size from a cherry to a hazel-nut. These are true aneurismal dilatations, and, according to Eppinger's careful study, consist of the intima and the adventitia, the elastic lamina having disappeared. The condition has been met with in children. Some of the cases, however, have been in adults; but the term as applied by Eppinger expresses, and probably correctly, the deep-seated fundamental error in development which must be at the basis of this condition. The coronary arteries is a favorite situation; a case has been reported by Gee in a boy of seven.

SECTION VI.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

I. ANÆMIA.

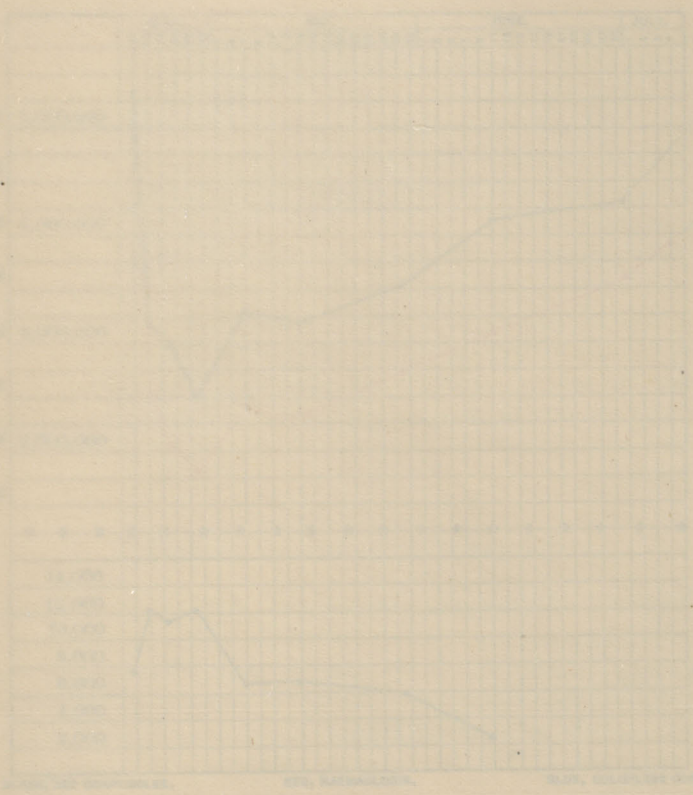
Anæmia may be defined as a reduction in the amount of the blood as a whole or of its corpuscles, or of certain of its more important constituents, such as albumen and hæmoglobin. The condition may be general or local. The former alone we are here considering. It is interesting to note, however, that the pallor, particularly of the face, which is one of the most striking symptoms of anæmia, is just as characteristic of local anæmia due to fright or to nausea. There are persons persistently pale without actual anæmia in whom the condition may be due to inherited peculiarities.

Our knowledge is not yet sufficiently advanced to classify satisfactorily the various forms of anæmia. The following provisional grouping may be made: (1) Secondary or symptomatic anæmia; (2) primary, essential, or cytogenic anæmia.

Secondary Anæmia.

Under this division comes a large proportion of all cases. The following are the most important groups, based on the etiology:

(1) *Anæmia from hæmorrhage*, either traumatic or spontaneous. The loss of blood may be rapid, as in lesions of large vessels, in injury or in rupture of aneurisms, or in cases of ulcer of the stomach or duodenum, or post-partum hæmorrhage. If the loss is excessive, death results from lowering of the arterial pressure. In sudden profuse hæmorrhage the loss of three or four pounds of blood may prove fatal. In the rupture of an aneurism into the pleura the loss of blood may amount to seven pounds and a half, the largest quantity I have known to be shed into one cavity. In a case of hæmatemesis the patient lost over ten pounds by measurement in one week and yet recovered from the immediate effects. Even after very severe hæmorrhage the number of red blood-corpuscles is not reduced so greatly as in forms of idiopathic anæmia. Thus in a case just



mentioned, at the termination of the week of bleeding there were nearly 1,390,000 red blood-corpuscles to the cubic millimetre. The process of regeneration goes on with great rapidity, and in some "bleeders" a week or ten days suffice to re-establish the normal amount. The watery and saline constituents of the blood are readily restored by absorption from the gastro-intestinal tract. The albuminous elements also are quickly renewed, but it may take weeks or months for the corpuscles to reach the

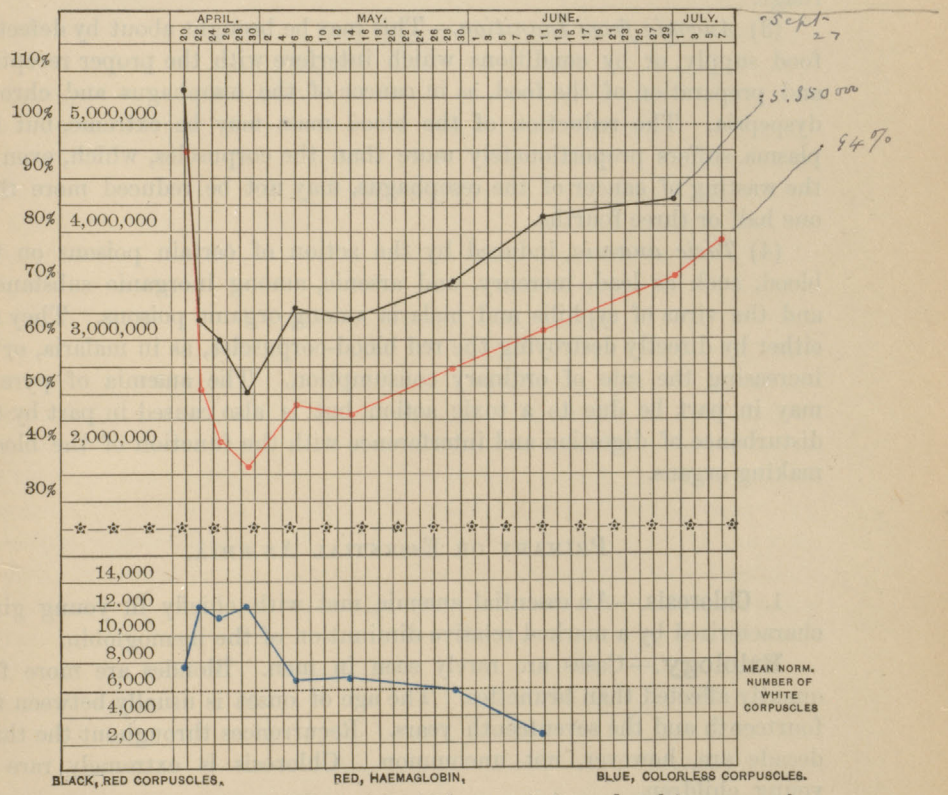


CHART XVI.—Illustrates the rapidity with which anæmia is produced in purpura hæmorrhagica and the gradual recovery.*

normal standard. The accompanying chart illustrates the rapid fall and gradual restitution in a case of severe purpura hæmorrhagica.

The microscopical characters of the blood after severe hæmorrhage are not much changed; the white corpuscles are relatively increased, producing a condition of leucocytosis. Nucleated red corpuscles are present, though usually not numerous. In the regeneration of the blood the de-

* On September 27th the patient returned from the country, where she had spent the summer. The blood count was then: Red corpuscles, 5,350,000; white corpuscles, 5,500; hæmoglobin, ninety-four per cent.

velopment of the hæmoglobin does not keep pace with that of the corpuscles.

(2) Anæmia is frequently produced by long-continued drain on the albuminous materials of the blood, as in chronic suppuration and Bright's disease. Prolonged lactation acts in the same way. Rapidly growing tumors may cause a profound anæmia, as in gastric cancer. The character of the blood in these cases is similar to that in anæmia after hæmorrhage.

(3) *Anæmia from Inanition.*—This may be brought about by defective food supply, or by conditions which interfere with the proper reception and preparation of the food, as in cancer of the œsophagus and chronic dyspepsia. The reduction of the blood mass may be extreme, but the plasma suffers proportionately more than the corpuscles, which, even in the wasting of cancer of the œsophagus, may not be reduced more than one half or three fourths.

(4) *Toxic anæmia*, induced by the action of certain poisons on the blood, such as lead, mercury, and arsenic, among inorganic substances, and the virus of syphilis and malaria among organic poisons. They act either by directly destroying the red blood-corpuscles, as in malaria, or by increasing the rate of ordinary consumption. The anæmia of pyrexia may in part be due to a toxic action, but is also caused in part by the disturbance of digestion and interference with the function of the blood-making organs.

Primary or Essential Anæmia.

1. **Chlorosis.**—An essential anæmia met with chiefly in young girls, characterized by a marked relative diminution of the hæmoglobin.

Etiology.—Cases are rarely seen in men. Blondes are more frequently affected than brunettes. The age of onset is usually between the fourteenth and the seventeenth years. Recurrences throughout the third decade are, however, not uncommon. Chlorosis is extremely rare in young children.

Hereditary influences probably play a part. Virchow pointed out that in many cases there was a defective development of the circulatory system, either congenital or resulting in a failure of the normal rate of growth. In some instances a compensatory hypertrophy of the heart has been found.

The disease is most common among the ill-fed, overworked girls of large towns, who are confined all day in close, badly-lighted rooms, or have to do much stair-climbing. Cases are frequent, however, under the most favorable conditions of life. Lack of proper exercise and fresh air, and improper food are important factors. Emotional and nervous disturbances may be prominent, so prominent that certain writers have regarded the disease as a neurosis. Menstrual disturbances are not un-

common, but are probably a sequence, not a cause of chlorosis. Sir Andrew Clark believes that constipation plays an important *rôle*, and that the condition is in reality a *coprœmia* due to the absorption of poisons—leucomaines and ptomaines—from the large bowel.

Morbid Anatomy.—Fortunately the disease is rarely fatal. The fat is well retained. Hypoplasia of the aorta and larger arteries has been found in some cases, and the vessels have had a remarkable degree of elasticity. The heart is usually dilated and the left ventricle hypertrophied. Hypoplasia of the uterus and defective development of the genitalia have also been found.

Symptoms.—The blood examination: Johann Duncan in 1867 first called attention to the fact that the essential feature was not a quantitative but a qualitative change in the hæmoglobin. This has been abundantly confirmed. The red blood-corpuscles may show only a moderate grade of reduction, but the corpuscles themselves are very poor in hæmoglobin. Thus in forty consecutive cases examined at my clinic by Thayer, the average number per cubic millimetre of the red blood-corpuscles was 4,225,181, or over eighty per cent, whereas the percentage of hæmoglobin for the total number was 44·1 per cent. The accompanying chart illustrates well these striking differences. The least blood-count in the series of cases referred to above was 1,932,000. There may be all the physical characteristics and symptoms of a profound anæmia with blood-corpuscles nearly at the normal standard. Thus in one instance the globular richness was over eighty-five per cent with the hæmoglobin about thirty-five. These characteristics are distinctive, I believe, and not found in the same grade in any other form of anæmia. The importance of the reduction in the hæmoglobin depends upon the fact that it is the iron-containing element of the blood with which in respiration the oxygen enters into combination. This marked diminution in the iron has also been determined by chemical analysis of the blood. The microscopical characteristics of the blood are as follows: In severe cases the corpuscles may be extremely irregular in size—poikilocytosis—which may occasionally be as marked as in pernicious anæmia. The large forms of red blood-cells are common, but the average size is stated to be below normal. The color of the corpuscles is noticeably pale and the deficiency may be seen either in individual corpuscles or in the blood mixture prepared for counting. The leucocytes may show a slight increase; thus, the average of the forty cases above referred to was 8,256 per cubic millimetre.

The general symptoms of chlorosis are those of an anæmia of moderate grade. The subcutaneous fat is well retained or even increased in amount. The complexion is peculiar; neither the blanched aspect of hæmorrhage nor the muddy pallor of grave anæmia, but a curious yellow-green tinge which has given the name to the disease, and to its popular designation, the green sickness. In cases of moderate grade the color may be deceptive, as the cheeks have a reddish tint, particularly on exertion (chlorosis

rubra). The subjects complain of breathlessness and palpitation, and there may be a tendency to fainting. The palpitation and breathlessness

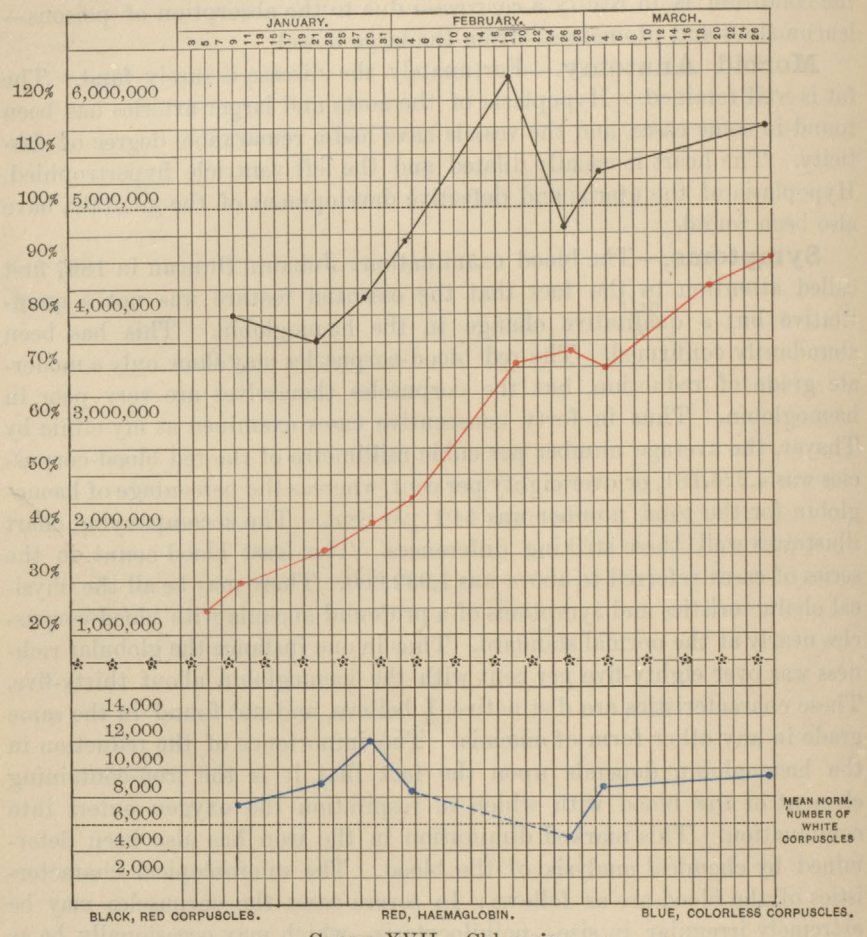


CHART XVII.—Chlorosis,

often lead to the suspicion of heart or lung disease. The eyes have a peculiar brilliancy and the sclerotics are of a sky-blue color Occasionally the skin shows areas of pigmentation, particularly about the joints.

Digestive symptoms are common. The appetite is capricious and the patients often have a longing for unusual articles, particularly acids. In some instances they eat all sorts of indigestible things, such as chalk or even earth. Constipation is a common symptom, and, as already mentioned, has been regarded as an important element in causing the disease. Contourier has noted the frequent association of dilatation of the stomach with chlorosis, and states that in some cases this may be an etiological factor, while in others it may be a result.

The circulatory symptoms are important. Palpitation of the heart oc-

cuss on exertion, and may be the most distressing symptom of which the patient complains. Percussion may show slight increase in the transverse dulness. A systolic murmur is heard at the apex or at the base, more commonly at the latter, but in extreme cases at both. A diastolic murmur is rarely heard. The systolic murmur is usually loudest in the second left intercostal space, where there is sometimes a distinct pulsation. The exact mode of production is still in dispute. Balfour holds that it is produced at the mitral orifice by relative insufficiency of the valves in the dilated condition of the ventricle. On the right side of the neck over the jugular vein a continuous murmur is heard, the *bruit de diable*, or humming-top murmur.

The pulse is usually full and soft. Pulsation in the peripheral veins is sometimes seen. There is a tendency to thrombosis in the veins, most commonly in the femoral, but in other instances in the longitudinal sinus, or the thrombosis may be multiple. Except in the sinuses, the condition is rarely serious. Tuckwell has reported an instance in which there was embolism of the right axillary artery with the loss of a thumb and part of the fingers. Brayton Ball has recently called attention to the importance of this feature of chlorosis.

As in all forms of essential anæmia, fever is not uncommon. Especial attention has of late been directed to this by French writers.* Chlorotic patients suffer frequently from headache and neuralgia, which may be paroxysmal. Hysterical manifestations are not infrequent. Menstrual disturbances are very common—amenorrhœa or dysmenorrhœa. With the improvement in the blood condition this function is usually restored.

Diagnosis.—The green sickness, as it is sometimes called, is in many instances recognized at a glance. The well-nourished condition of the girl, the peculiar complexion, which is most marked in brunettes, and the white sclerotics are very characteristic. A special danger exists in mistaking the anæmia of the early stage of pulmonary tuberculosis for chlorosis. The palpitation of the heart and shortness of breath frequently suggest heart-disease, and the œdema of the feet and general pallor cause the cases to be mistaken for Bright's disease. In the great majority of cases the characters of the blood readily separate chlorosis from other forms of anæmia.

2. **Idiopathic or Progressive Pernicious Anæmia.**—The disease was first clearly described by Addison, who called it idiopathic anæmia. Channing and Gusserow described the cases occurring post partum, but to Biermer we owe a revival of interest in the subject.

Etiology.—The existence of a separate disease worthy of the term progressive pernicious anæmia has been doubted, but there are unquestionably cases in which, as Addison says, there exist none of the usual causes or concomitants of anæmia. Clinically there are several different

* Traité, Paris Thesis, 1888.

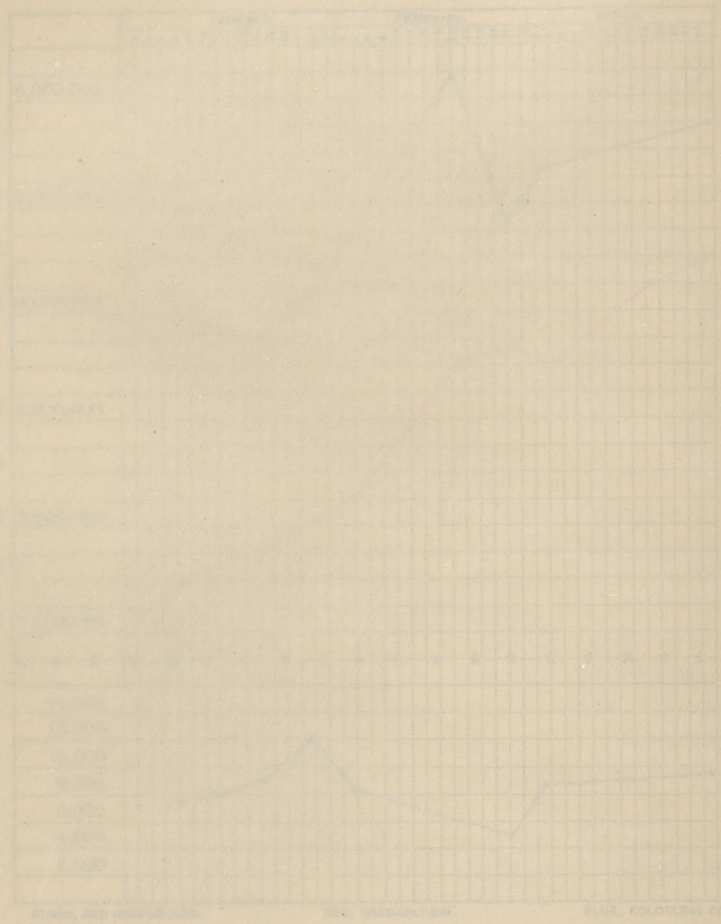


CHART XVII.—Chlorosis.

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* Trazit, Paris Thesis, 1888.

groups which present the characters of a progressive and pernicious anæmia and are etiologically different. Thus, a fatal anæmia may be due to the presence of parasites, or may follow hæmorrhage, or be associated with chronic atrophy of the stomach; but when we have excluded all these causes there remains a group which, in the words of Addison, is characterized by a "general anæmia occurring without any discoverable cause whatever, cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease."

Idiopathic anæmia is widely distributed. It is of frequent occurrence in the Swiss Cantons, and is not uncommon in this country. It affects middle-aged persons, but instances in children have been described. Griffith mentions about ten cases occurring under twelve years of age. The youngest patient I have seen was a girl of twenty. Males are more frequently affected than females. Of my 27 cases, 10 were females and 17 were males. Of 110 cases collected by Coupland, 56 were in men and 54 in women.

With the following conditions may be associated a profound anæmia not to be distinguished clinically from Addison's idiopathic form:

(*a*) *Pregnancy and Parturition.*—The symptoms may develop during pregnancy, as in 19 of 29 cases of this group in Eichhorst's table. More commonly, in my experience, the condition has been post partum; thus, of my 27 cases, 5 followed delivery.

(*b*) *Atrophy of the Stomach.*—This condition, early recognized by Flint and Fenwick, may certainly cause a progressive pernicious anæmia. By modern methods it may now be possible to exclude this extreme gastric atrophy.

(*c*) *Parasites.*—The most severe form may be due to the presence of parasites, and the accounts of cases depending upon the anchylostoma and the bothriocephalus describe a progressive and often pernicious anæmia.

After the exclusion of these forms there remains a large proportion, numbering eighteen cases in my series, which correspond to Addison's description. The etiology of these cases is still dark. The researches of Quincke and his student Peters showed that there was an enormous increase in the iron in the liver, and he suggested that the affection was probably due to increased hæmolysis. This has been strongly supported by the extensive observations of Hunter, who has also shown that the urine excreted is darker in color and contains pathological urobilin. The lemon tint of the skin or the actual jaundice is attributed, on this view, to the changes in the liver cells produced by the excessive amount of pigment, but in the light grades it is unquestionably hæmatogenous. To explain the hæmolysis, it has been thought that in the condition of faulty gastro-intestinal digestion, which is so commonly associated with these cases, poisonous materials are developed, which when absorbed cause destruction of the corpuscles. Certainly the evidence for hæmolysis is very

strong, but we are still far away from a full knowledge of the conditions under which it is produced.

On the other hand, F. P. Henry, Stephen Mackenzie, and other authorities incline to the belief that the essence of the disease is in defective hæmogenesis, in consequence of which the red blood-corpuscles are abnormally vulnerable. A point noted by Copeman, that the hæmoglobin crystallizes from the blood-corpuscles with great readiness, can scarcely be regarded as favoring the view of imperfect hæmogenesis, since this is a feature specially characteristic of the blood of the young.

Morbid Anatomy.—The body is rarely emaciated. A lemon tint of the skin is present in a majority of the cases. The muscles often are intensely red in color, like horse-flesh, while the fat is light yellow. Hæmorrhages are common on the skin and serous surfaces. The heart is usually large, flabby, and empty. In one instance I obtained only two drachms of blood from the right heart, and between three and four from the left. The muscle substance of the heart is intensely fatty, and of a pale, light-yellow color. In no affection do we see more extreme fatty degeneration. The lungs show no special changes. The stomach in many instances is normal, but in some cases of fatal anæmia the mucosa has been extensively atrophied. In the case described by Henry and myself the mucous membrane had a smooth, cuticular appearance, and there was complete atrophy of the secreting tubules. The liver may be enlarged and fatty. In most of my autopsies it was normal in size, but usually fatty. The iron is in excess, and in striking contrast to cases of secondary anæmia. It is deposited in the outer and middle zones of the lobules, and in two specimens which I examined seemed to have such a distribution that the bile capillaries were distinctly outlined. This is certainly, as Hunter states, a special and characteristic lesion, possibly peculiar to pernicious anæmia. A. J. Scott examined for me the livers in forty-five consecutive autopsies without finding (except in pernicious anæmia) this special distribution of pigment.

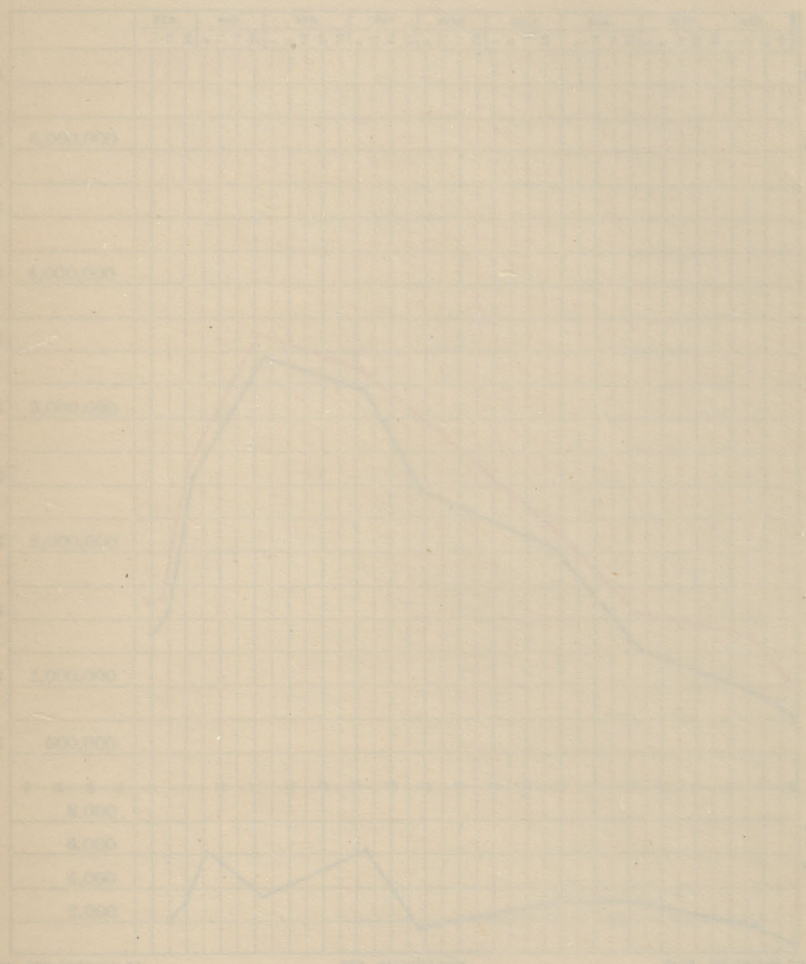
The spleen shows no important changes. In one of Palmer Howard's cases the organ weighed only an ounce and five drachms. The iron pigment is usually in excess. The lymph glands may be of a deep red color. The amount of iron pigment is increased in the kidneys, chiefly in the convoluted tubules. The bone marrow, as pointed out by H. C. Wood, may resemble that of a child. This observation has been repeatedly confirmed, but the condition does not appear to be constant. Changes in the ganglion cells of the sympathetic have been reported on several occasions. Lichtheim has found sclerosis in the posterior columns of the cord, which he thinks secondary to the anæmia, and a similar change has been met with in two recent cases by Morris Lewis and Burr.

Symptoms.—The patient may have been in previous good health, but in many cases there is a history of gastro-intestinal disturbance, mental shock, or worry. The description given by Addison presents the chief

features of the disease in a masterly manner. "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

The Blood.—The corpuscles may sink to one fifth or less of the normal number. They may sink to 500,000 per cubic millimetre, and in a case of Quincke's the number was reduced to 143,000 per cubic millimetre. The hæmoglobin is relatively increased, so that the individual globular richness is plus, a condition exactly the opposite to that which occurs in chlorosis, in which the corpuscular richness in coloring matter is minus. The relative increase in the hæmoglobin is probably associated with the average increase in the size of the red blood-corpuscles. The accompanying chart illustrates these points. Microscopically the red blood-corpuscles present a great variation in size, and there can be seen large giant forms, megalocytes, which are often ovoid in form, measuring eight, eleven, or even fifteen micromillimetres in diameter, a circumstance which Henry regards as indicating a reversion to a lower type. Laache thinks these pathognomonic, and they certainly form a constant feature. There are also small round cells, microcytes, from two to six micromillimetres in diameter, and of a deep red color. The corpuscles show a remarkable irregularity in form, elongated and rodlike or pyriform; one end of a corpuscle may retain its shape while the other is narrow and extended. To this condition of irregularity Quincke gave the name poikilocytosis. The leucocytes are generally diminished in number, and the relative percentage of the mononuclear elements is somewhat higher than in normal blood.

Nucleated red blood-corpuscles are constantly present, as pointed out by Ehrlich. Besides the ordinary form, which is of the same size as the common corpuscle and which has a small, deeply stained nucleus (normo-



blasts), there are very large forms with large, palely staining nuclei (gigantoblasts), which resemble somewhat the larger megalocytes. Ehrlich re-

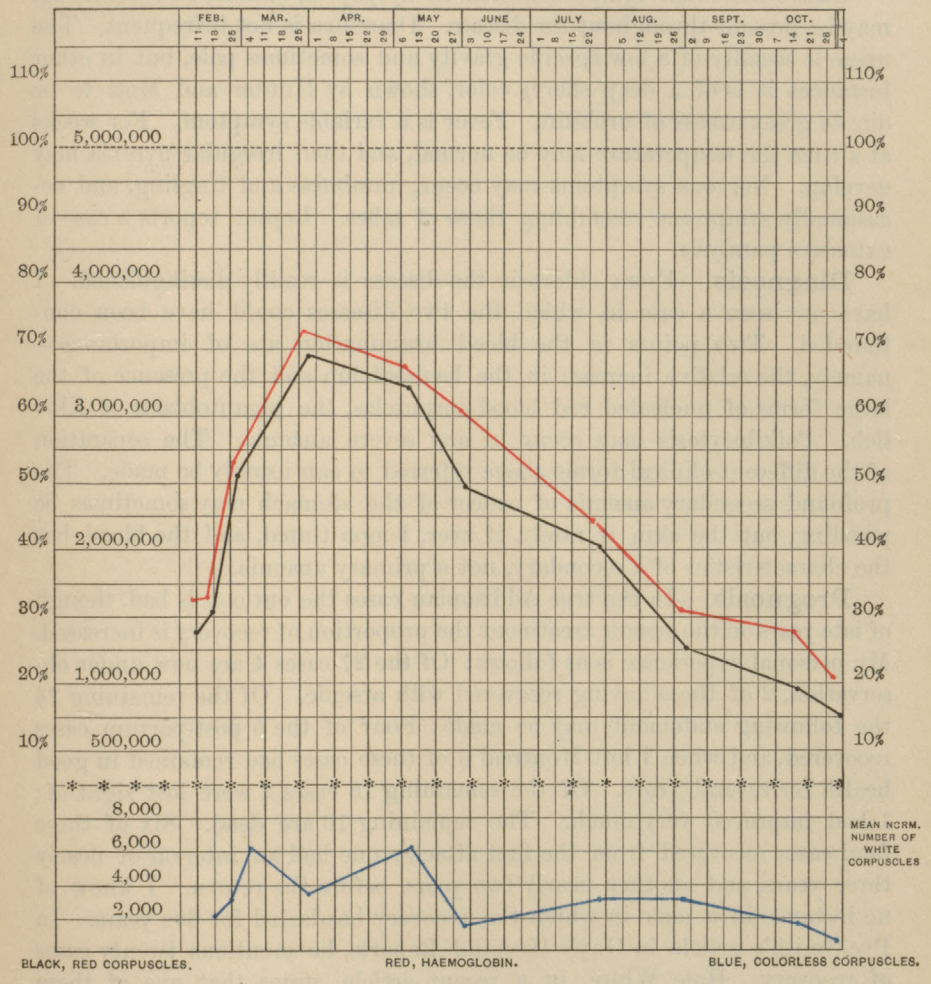


CHART XVIII.—Pernicious anæmia.

gards the presence of these as almost distinctive of progressive pernicious anæmia; they are only found here and in the later stages of leukæmia. The blood-plates are either absent or very scanty.

The cardio-vascular symptoms are important and are noted in the description given above. Hæmic murmurs are constantly present. The larger arteries pulsate visibly and the throbbing in them may be distressing to the patient. The pulse is full and frequently suggests the water-hammer beat of aortic insufficiency. The capillary pulse is frequently to be seen. The superficial veins are often prominent, and in two cases I have seen well-marked pulsation in them. Hæmorrhages may occur, either

in the skin or from the mucous surfaces. Retinal hæmorrhages are common. There are rarely symptoms in the respiratory organs.

Gastro-intestinal symptoms, such as dyspepsia, nausea, and vomiting, may be present throughout the disease. Diarrhœa is not infrequent. The urine is usually of a low specific gravity and sometimes pale, but in other instances it is of a deep sherry color, shown by Hunter and Mott to be due to great excess of urobilin. Fever is a variable symptom. For weeks at a time the temperature may be normal, and then irregular pyrexia may develop. Nervous symptoms may occur, numbness and tingling, and occasionally symptoms resembling those of tabes. Lepine reports a case of extensive paralysis.

Diagnosis.—From chlorosis the disease is readily distinguished. I have not seen a case in which the two diseases could have been confounded. Two points in the blood examination are of importance—namely, the relative increase in the hæmoglobin and the presence of the large forms of nucleated red blood-corpuscles, the gigantoblasts of Ehrlich. Poikilocytosis may occur in any severe anæmia. The separation of the different clinical forms above referred to can usually be made. The profound secondary anæmia of cancer of the stomach may sometimes be puzzling, but the skin is rarely, if ever, lemon-tinted, and the blood has the characteristics of a secondary, not a primary anæmia.

Prognosis.—In the true Addisonian cases the outlook is bad, though of late years on the arsenic treatment the proportion of recovery is increased. My personal experience is as follows: Of the 27 cases 4 are now under observation, 2 of these having recovered with arsenic. Of the remaining 23 the following statement may be made: Four of the 5 post-partum cases recovered, and when I left Montreal 3 of these cases had remained in good health for several years. Of the remaining 18 cases 2 were lost sight of; 1 had improved very much. The remaining 16 are dead. Six of these fatal cases recovered from the first attack; one had an interval of nearly three years, and another nearly two years, before the return. I know of no instance in a male in which the recovery has lasted for five years. In Pye-Smith's article in Guy's Hospital Reports, he mentions twenty cases of recovery. Hale White, in a recent article, states that one of these cases, treated by arsenic in 1880, remained alive and well January, 1891. One of my patients made an apparently complete recovery and resumed active business and political duties. So characteristic are recurrences in this affection that Stephen Mackenzie, in his recent lectures, considered them under a separate heading of relapsing pernicious anæmia.

Treatment of Anæmia.—*Secondary Anæmia.*—The traumatic cases do best, and with plenty of good food and fresh air the blood is readily restored. The extraordinary rapidity with which the normal percentage of red blood-corpuscles is reached without any medication whatever is an important lesson. The cause of the hæmorrhage should be sought and the necessary indications met. The large group depending

J. Blaud. Mémoir sur les maladies de Beaucaire chlorotiques. Rev. Med. 1832 T. I p 237

on the drain on the albuminous materials of the blood, as in Bright's disease, suppuration, and fever, is difficult to treat successfully, and so long as the cause keeps up it is impossible to restore the normal blood condition. The anæmia of inanition requires plenty of nourishing food. When dependent on organic changes in the gastro-intestinal mucosa not much can be expected from either food or medicine. In the toxic cases due to mercury and lead, the poison must be eliminated and a nutritious diet given with full doses of iron. In a great majority of these cases there is deficient blood formation, and the indications are briefly three—plenty of food, an open-air life, and iron. As a rule it makes but little difference what form of the drug is administered.

The treatment of *chlorosis* affords one of the most brilliant instances—of which we have but three or four—of the specific action of a remedy. Apart from the action of quinine in malarial fever, and of mercury and iodide of potassium in syphilis, there is no other remedy the beneficial effects of which we can trace with the accuracy of a scientific experiment. It is a minor matter *how* the iron cures chlorosis. In a week we give to a case as much iron as is contained in the entire blood, as even in the worst case of chlorosis there is rarely more than a deficit of two grammes of this metal. Iron is present in the fæces of chlorotic patients before they are placed upon any treatment, so that the disease does not result from any deficiency of available iron in the food. Bunge believes that it is the sulphur which interferes with the digestion and assimilation of this natural iron. The sulphides are produced in the process of fermentation and decomposition in the fæces, and interfere with the assimilation of the normal iron contained in the food. By the administration of an inorganic preparation of iron with which these sulphides combine the natural organic combinations in the food are spared. In studying a number of charts of chlorosis it is seen that there is an increase in the red blood-corpuscles under the influence of the iron, and in some instances the globular richness rises above normal. The increase in the hæmoglobin is slower and the maximum percentage may not be reached for a long time. I have for years in the treatment of chlorosis used with the greatest success Blaud's pills, made and given according to the formula in Niemeyer's text-book, in which each pill contains 2 grs. of the sulphate of iron. During the first week one pill is given three times a day. In the second week, two pills; in the third week, three pills, three times a day. This dose should be continued for four or five weeks, at least, before reduction. An important feature in the treatment of chlorosis is to persist in the use of the iron for at least three months, and if necessary subsequently to resume it in smaller doses, as recurrences are so common. The diet should consist of good, easily digested food. Special care should be directed to the bowels, and if constipation is present a saline purge should be given each morning. Such stress does Sir Andrew Clark lay on the importance of constipation in chlorosis that he states that if limited to the choice of one drug in the treatment of the

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disease he would choose a purgative. Dilute hydrochloric acid, manganese, phosphorus, and oxygen have been recommended.

Treatment of Pernicious Anæmia.—Since the introduction by Byrom Bramwell of arsenic in this affection a large number of cases have been temporarily, a few permanently, cured by it. It should be given as Fowler's solution in increasing doses. It is usually well borne, and patients, as a rule, take up to twenty minims three times a day without any disturbance. I usually begin with three minims and increase to five at the end of the first week, to ten at the end of the second week, to fifteen at the end of the third week, and, if necessary, increase to twenty or twenty-five. In a case in which the recovery persisted for nearly three years, the dose was gradually increased to thirty minims. These patients seem to bear the arsenic extremely well. It is sometimes better borne as arsenious acid in pill form. Vomiting and diarrhœa are rare; occasionally puffiness of the face is produced, and in some cases pigmentation of the skin.

Rest in bed and a light but nutritious diet (giving the food in small amounts and at fixed intervals) are the first indications. I always prefer to begin the treatment of a case of pernicious anæmia, whatever the grade may be, with rest in bed as one of the essential elements. Massage will also be found very beneficial. I have abandoned the use of rectal injections of dried blood. Iron seems to have no action in this form, but in a case in which the arsenic disagrees it may be tried.

II. LEUKÆMIA.

Definition.—An affection characterized by persistent increase in the white blood-corpuscles, associated with enlargement, either alone or together, of the spleen, lymphatic glands, or bone marrow.

The disease was described almost simultaneously by Virchow and by Bennett, who gave to it the name leucocythæmia.

Etiology.—We know nothing of the conditions under which the disease develops. It is not uncommon on this continent. Of 17 cases of which I have notes, 11 occurred in Montreal, 2 in Philadelphia, and 4 within the past two years at the Johns Hopkins Hospital. It does not seem more frequent in the southern parts of the country.

The disease is most common in the middle period of life. The youngest of my cases was a child of eight months, and cases are on record of the disease as early as the eighth or tenth week. It may occur as late as the seventieth year. Males are more prone to the affection than females. Of my cases 11 were in males and 6 in females. Birch-Hirschfeld states that of 200 cases collected from the literature, 135 were males and 65 females.

A tendency to hæmorrhage has been noted in many cases, and some of the patients have suffered repeatedly from nose-bleeding. In women

the disease is most common at the climacteric. There are instances in which it has developed during pregnancy. The case described by J. Chalmers Cameron, of Montreal, is in this respect remarkable, as the patient passed through three pregnancies, bearing on each occasion non-leukæmic children. The case is interesting, too, as showing the hereditary character of the affection, as the grandmother and mother, as well as a brother, suffered from symptoms strongly suggestive of leukæmia. One of the patient's children had leukæmia before the mother showed any signs, and a second died of the disease. At the last report this patient had gradually recovered from the third confinement and the red blood-corpuscles had risen to 4,000,000 per cubic millimetre, and the ratio of white to red 1 to 200. Sänger has reported a case in which a healthy mother bore a leukæmic child.

Malaria is believed by some to be an etiological factor. Of 150 cases analyzed by Gowers, there was a history of malaria in 30; in my series there was a history in at least 7. Syphilis appears in some cases to have been closely associated with the disease. The disease has followed injury or a blow.

The lower animals are subject to the affection, and cases have been described in horses, dogs, oxen, cats, swine, and mice.

Morbid Anatomy.—The wasting may be extreme, and dropsy is sometimes present. There is in many cases a remarkable condition of polyæmia; the heart and veins are distended with large blood-clots. In Case XI of my series the weight of blood in the heart chambers alone was 620 grammes. There may be remarkable distention of the portal, cerebral, pulmonary, and subcutaneous veins. The blood is usually clotted, and the enormous increase in the leucocytes gives a pus-like appearance to the coagula, so that it has happened more than once, as in Virchow's memorable case, that on opening the right auricle the observer at first thought he had cut into an abscess. The coagula have a peculiar greenish color, somewhat like the fat of a turtle. The alkalinity of the blood is diminished. The fibrin is increased. The character of the corpuscles will be described under the symptoms. Charcot's octohedral crystals separate from the blood after death. The specific gravity of the blood is somewhat lowered. There may be pericardial ecchymoses.

The spleen in the great majority of cases is enlarged. Strong adhesions may unite it to the abdominal wall, the diaphragm, or the stomach. The capsule may be thickened. The vessels at the hilus are enlarged; the weight may range from two to eighteen pounds. The organ is in a condition of chronic hyperplasia. It cuts with resistance, has a uniformly reddish-brown color, and the Malpighian bodies are invisible. Grayish-white, circumscribed, lymphoid tumors may occur throughout the organ, contrasting strongly with the reddish-brown matrix. In the early stage the swollen spleen pulp is softer, and it is stated that rupture has occurred from the intense hyperæmia. Enlargement of the lymphatic glands may

occur, either in conjunction with splenic enlargement or alone. In only one of my cases was the enlargement notable. In the cases of lymphatic leukæmia the cervical, axillary, mesenteric, and inguinal groups may be much enlarged, but the glands are usually soft, isolated, and movable. They may vary considerably in size during the course of the disease. The tonsils and the lymph follicles of the tongue, pharynx, and mouth may be enlarged.

In the majority of cases the bone marrow is involved with the spleen, the lieno-medullary form of the Germans. The marrow may be involved alone, forming a pure myelogenous leukæmia. Instead of a fatty marrow, the medulla of the long bones may resemble the consistent matter which forms the core of an abscess, or it may be dark brown in color. In Ponfick's case there were hæmorrhagic infarctions. There may be much expansion of the shell of bone and localized swellings which are tender and may even yield to firm pressure. Histologically, there are found in the medulla large numbers of nucleated red corpuscles in all stages of development, numerous cells with eosinophilic granules, and also many cells corresponding to the *myelocytes* found in the blood. Large mononuclear cells in the process of division by karyokinesis may be abundant. Polynuclear leucocytes are also present, as well as a certain number of small mononuclear elements.

The thymus is rarely involved, though it has been enlarged in some of the cases of acute lymphatic leukæmia.

In a few instances there have been leukæmic enlargements in the solitary and agminated glands of Peyer. In a case of Willcocks there were growths on the surface of the stomach and gastro-splenic omentum.

The liver may be enlarged, and in a case described by Welch it weighed over thirteen pounds. The enlargement is usually due to a diffuse leukæmic infiltration. The columns of liver cells are widely separated by leucocytes, which are partly within and partly outside the lobular capillaries. There may be definite leukæmic growths.

There are rarely changes of importance in the lungs. The kidneys are often enlarged and pale, the capillaries may be distended with leucocytes, and leukæmic tumors may occur. The skin may be involved, as in a case described by Kaposi.

Leukæmic tumors in the organs are not common. They were present in only one of the twelve autopsies in my series. In 159 cases collected by Gowers there were only thirteen instances of leukæmic nodules in the liver and ten in the kidneys. These new growths probably develop from leucocytes which leave the capillaries. Bizzozero has shown that the cells which compose them are in active fission.

Symptoms.—The onset is insidious, and, as a rule, the patient seeks advice for progressive enlargement of the abdomen and shortness of breath, or for the enlarged glands or the pallor, palpitation, and other symptoms of anæmia. Bleeding at the nose is common. Gastro-intestinal

Anæmia is not a necessary accompaniment of the disease – this noted

symptoms may precede the onset. Occasionally the first symptoms are of a very serious nature. In one of the cases of my series the boy played lacrosse two days before the onset of the final hæmatemesis, and in another case, a girl who had, it was supposed, only a slight chlorosis, died of fatal hæmorrhage from the stomach before any suspicion had been aroused as to the true condition.

Blood.—In all forms of the disease the diagnosis must be made by the examination of the blood, as it alone offers distinctive features. In the normal blood Ehrlich recognizes the following varieties of colorless elements: (*a*) Lymphocytes—small cells about the size of a red blood-corpuscle, and probably derived from the lymphatic glands, which have a single large, round, deeply staining nucleus, surrounded by a narrow rim of non-granular protoplasm. (*b*) Large mononuclear leucocytes—cells several times as large as the red blood-corpuscle, with an oval or elliptical nucleus and a relatively larger amount of ungranulated protoplasm. (*c*) Transitional forms—cells which resemble the last variety, but have indentations and irregularities in the nucleus. (*d*) Polynuclear leucocytes—these are about the same size or a little smaller than the last variety. The nucleus is a long, deeply staining body which is bent and twisted on itself into irregular shapes. The protoplasm of these cells is filled with granules, which are stained, not by acid or basic coloring matters alone, but only by a combined fluid. The granules are therefore termed neutrophilic, and the name "neutrophiles" is given to these cells. (*e*) Cells about the same size as the last, but containing large, highly refractile, fat-like granules, which have an affinity for acid coloring matters. On account of their affinity for eosin, Ehrlich terms them *eosinophiles*. In normal blood these cells occur in a definite proportion to each other; the lymphocytes fifteen to thirty per cent, the polynuclear sixty-five to eighty per cent, the mononuclear and transitional forms about six per cent, and the eosinophiles two to four per cent.

The most striking change in the more common form, the lieno-myelogenic, is the increase in the colorless corpuscles. The average number of white per cubic millimetre is estimated at about 6,000; thus the proportion of white to red is 1 to 500—1,000. In leukæmia the proportion may be 1 to 10, or 1 to 5, or the ratio may reach 1 to 1. There are instances on record in which the number of leucocytes has exceeded that of the red corpuscles.

The character of the cells in splenic myelogenous leukæmia is as follows: The lymphocytes are little, if at all, increased; relatively they are greatly diminished. The eosinophiles are present in normal or increased relative proportion, so that there is a great total increase, and their presence is a striking feature in the stained blood-slide. The polynuclear neutrophiles may be in normal proportion; more frequently they are relatively diminished, and in the latter stages they may form but a small proportion of the colorless elements. The most characteristic feature

of the blood in this form of leukæmia is the presence of cells which do not occur in normal blood. They appear to be derived from the marrow, and are called by Ehrlich *myelocytes*. They are as large or even larger than the large mononuclear leucocytes, and are similar to them in appearance, but differ from them in the fact that the protoplasm is filled with the fine neutrophilic granules. Müller has recently found many large mononuclear elements with karyokinetic figures in leukæmic blood and in the marrow.

Nucleated red blood-corpuscles are present, usually in considerable numbers. There is, as a rule, only a moderate reduction in the number of red blood-corpuscles, rarely under two million per cubic millimetre. The hæmoglobin is usually reduced in a somewhat greater proportion. The accompanying blood chart is from a case of leukæmia with an enormously enlarged spleen.

The histological characters of the blood in acute lymphatic leukæmia differ materially. The increase in the colorless elements is never so great as in the preceding form; a proportion of one to ten would be extreme. This increase takes place solely in the lymphocytes, all other forms of leucocytes being present in greatly diminished relative proportion. In Uthemann's case ninety-three per cent of all the leucocytes were lymphocytes. Eosinophiles and nucleated red corpuscles are rare. Myelocytes are not present. As occasionally combined forms of leukæmia may occur, so undoubtedly variations from these two types of blood may be met with, and in a case of acute leukæmia observed at the Johns Hopkins Hospital, in which glands, marrow, and spleen were affected, there was present, besides a large proportion of lymphocytes and myelocytes, a considerable number of large mononuclear leucocytes. Among other points about leukæmic blood may be mentioned the feebleness of the amœboid movement, as noted by Cafaſy, which may be accounted for by the large number of mononuclear elements present, the polynuclear alone possessing this power. The blood-plates exist in variable numbers; they may be remarkably abundant. The fibrin network between the corpuscles is usually thick and dense. In blood-slides which are kept for a short time, Charcot's octohedral crystals separate, and in the blood of leukæmia the hæmoglobin shows a remarkable tendency to crystallize.

The pulse is usually rapid, soft, compressible, but often full in volume. There are rarely any cardiac symptoms. The apex beat may be lifted an interspace by the enlarged spleen. Toward the close, as a consequence of the feeble circulation, œdema may occur in the feet or there may be general anasarca. Hæmorrhage is a common symptom and may be either late or early. Epistaxis is the most frequent form. Hæmoptysis and hæmaturia are rare. Bleeding from the gums may be present. Hæmatemesis proved fatal in two of my cases, and in a third a large cerebral hæmorrhage rapidly killed. The leukæmic retinitis is a part of the hæmorrhagic manifestations.

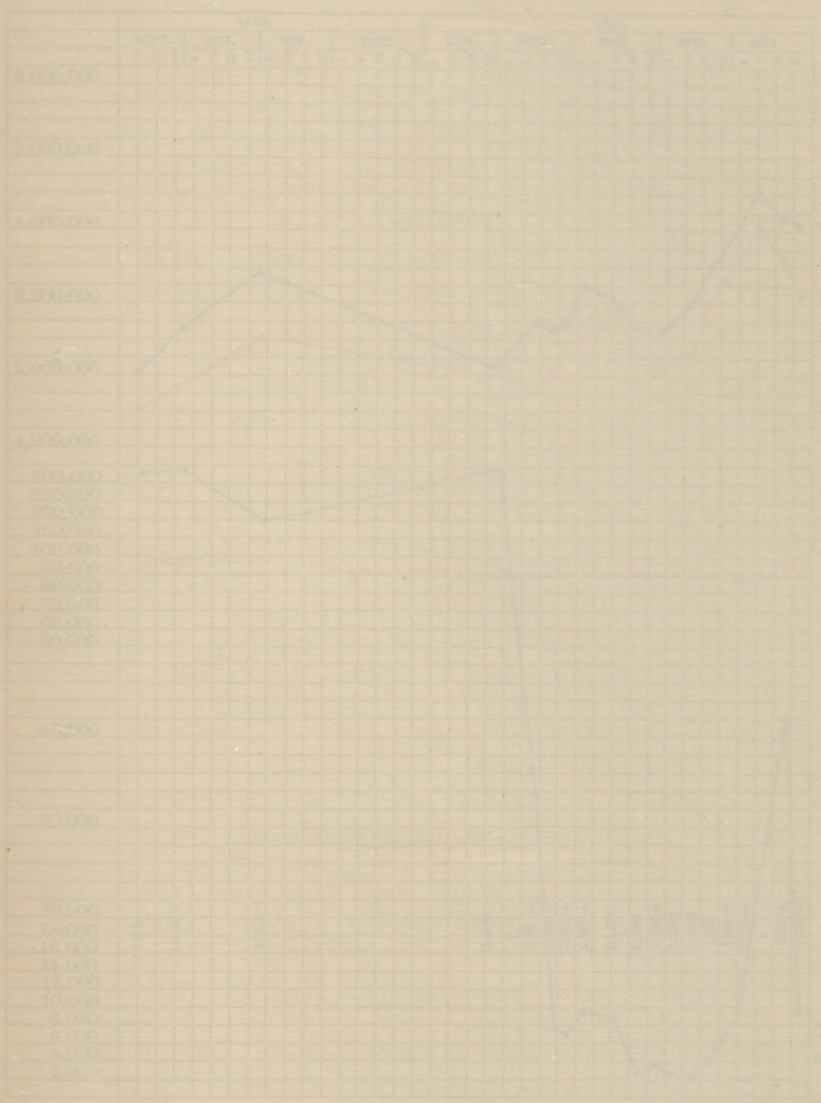


CHART XIX.—Leukæmia.

There are very few pulmonary symptoms. The shortness of breath is due, as a rule, to the anæmia. Toward the end there may be œdema of

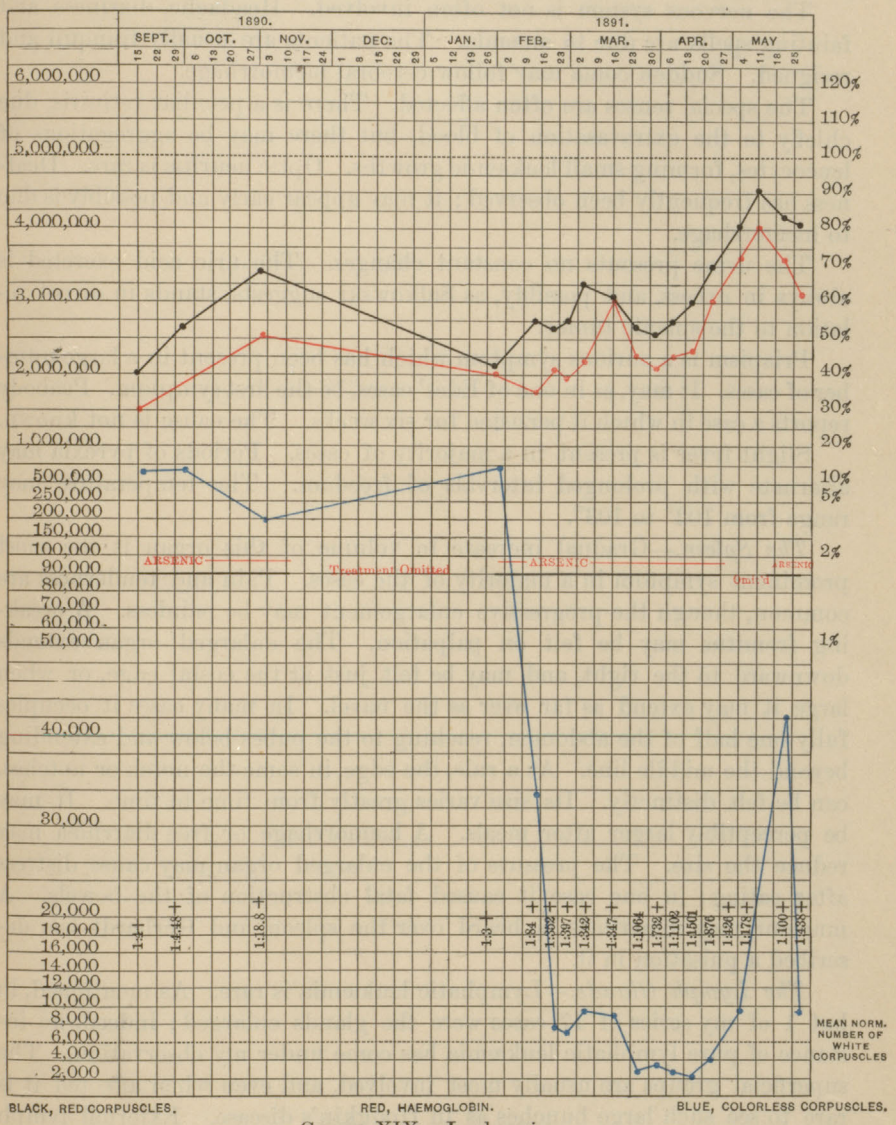


CHART XIX.—Leukæmia.

the lungs or pneumonia may carry off the patient. The gastro-intestinal symptoms are rarely absent. Nausea and vomiting are early features in some cases. Diarrhœa may be very troublesome, even fatal. Intestinal hæmorrhage is not common. There may be a dysenteric process in the colon. Jaundice rarely occurs, though in one case of my series there were recurrent attacks. Ascites may be a prominent symptom, probably due

to the presence of the splenic tumor. A leukæmic peritonitis also may be present, due to new growths in the membranes.

The nervous system is not often involved. Headache, dizziness, and fainting spells are due to anæmia. The patients are usually tranquil and resigned. Sudden coma may follow cerebral hæmorrhage.

The special senses are often affected. There is a peculiar retinitis, due chiefly to the extravasation of blood, but there may be aggregations of leucocytes, forming small leukæmic growths. Optic neuritis is rare. Deafness has frequently been observed; it may appear early and possibly is due to hæmorrhage.

The urine presents no constant changes. The uric acid excreted is always in excess, and possibly, as Salkowski suggests, stands in direct relation to the splenic tumor.

Priapism is a curious symptom which has been present in a large number of cases. It may, as in one of Edes' cases, be the first symptom. Peabody reports a case in which it persisted for six weeks. The cause is not known.

Slight fever is present in a majority of cases. Periods of pyrexia may alternate with prolonged intervals of freedom. The temperature may range from 102° to 103°.

The Spleen.—Gradual increase in volume of this organ is the most prominent symptom in a majority of the cases. Pain and tenderness are common, though the progressive enlargement may be painless. A creaking fremitus may be felt on palpation. The enlarged organ extends downward to the right, and may be felt just at the costal edge, or when large it may extend as far over as the navel. In many cases it occupies fully one half of the abdomen, reaching to the pubes below and extending beyond the middle line. As a rule, the edge, in some the notch or notches, can be felt distinctly. Its size varies greatly from time to time. It may be perceptibly larger after meals. A hæmorrhage or free diarrhœa may reduce the size. The pressure of the enlarged organ may cause distress after eating; in one case it caused fatal obstruction of the bowels. A murmur may sometimes be heard over the spleen, and Gerhardt has described a pulsation in it.

The Lymph Glands.—Lymphatic leukæmia is rare. As mentioned, in but 1 of my series of 17 cases were the glands enlarged; indeed, no instance of pure lymphatic leukæmia has come under my observation. The superficial groups are usually most involved, and even when affected it is rare to see such large bunches as in Hodgkin's disease. External lymph tumors are rare.

The pure myelogenous cases without associated enlargement of the spleen are rare. The most extreme hyperplasia of the bone marrow may exist without any tenderness. Occasionally the sternum, ribs, and flat bones show great irregularity and deformity, owing to definite tumor-like expansions.

Diagnosis.—The recognition of leukæmia can be determined only

There are however cases of extraordinary chronicity. In a case which I saw last year 1894, the diagnosis of lymphatic leukæmia was made by Wyatt Draper 8 years before & the glandular tumors had been persistent. The patients general co

by microscopical examination of the blood. The clinical features may be identical with those of ordinary splenic anæmia, or with Hodgkin's disease An interesting question arises whether real increase in the leucocytes is the only criterion of the existence of the disease. Thus, for instance, in the case whose chart is given, on page 701, the patient came under observation in September, 1890, with 2,000,000 red blood-corpuscles per cubic millimetre, thirty per cent of hæmoglobin, and 500,000 white blood-corpuscles per cubic millimetre—a proportion of one to four. As shown by the chart, throughout September, October, November, and December, this ratio was maintained. Early in January, under treatment with arsenic, the white corpuscles began to decrease and gradually, as shown in the chart, the normal ratio was reached. At this time could it be said that the case was one of leukæmia without increase in the number of leucocytes? The blood examination by Ehrlich's method, as made by Thayer, showed that the characteristic myelocytes, elements which are not present in normal blood, were still present in numbers sufficient, at any rate, to suggest, if the patient had come under observation for the first time, that leukæmia might occur. By Ehrlich's method of blood examination a condition of leucocytosis can readily be distinguished from that of leukæmia, for in all ordinary leucocytoses the increase takes place solely in the polynuclear neutrophilic leucocytes, forming quite a different picture from the characteristic conditions described above.

Prognosis.—Recovery occasionally occurs. A great majority of the cases prove fatal within two or three years. Unfavorable signs are a tendency to hæmorrhage, persistent diarrhœa, early dropsy, and high fever. Remarkable variations are displayed in the course, and a transient improvement may take place for weeks or even months. The pure lymphatic form seems to be of particular malignancy, some cases proving fatal in from six to eight weeks.

Treatment.—Fresh air, good diet, and abstention from mental worry and care, are the important general indications. The *indicatio morbi* cannot be met. There are certain remedies which have an influence upon the disease. Of these, arsenic, given in large doses, is the best. I have repeatedly seen improvement under its use. On the other hand, there are curious remissions in the disease which render therapeutical deductions very fallacious. I have seen such marked improvement without special treatment that the patient, from a bed-ridden, wretched condition, recovered strength enough to enable him to attend to light duties.

Quinine may be given in cases with a malarial history. Iron may be of value in some cases, as may also inhalations of oxygen.

Excision of the leukæmic spleen has been performed twenty-four times, with one recovery—the case of Franzolini. Fussell gives the statistics of 105 cases of splenectomy with 48 deaths. Of the cases of simple hypertrophy, 28 in number, 9 recovered. Of 16 cases of floating spleen, 15 recovered.

III. HODGKIN'S DISEASE.

Definition.—An affection characterized by progressive hyperplasia of the lymph glands, with anæmia, and occasionally the development of secondary lymphoid growths in liver, spleen, and other organs. The disease has also the names *pseudo-leukæmia*, *general lymphadenoma*, and *adénie*.

Hodgkin, the well-known morbid anatomist of Guy's Hospital, first described cases in detail, and by the labors of Wilks, Virchow, Billroth, and Cohnheim the disease attained definite recognition.

Etiology.—A majority of the cases are in young persons. In Gowers' table of 100 cases, 30 were under twenty years, 34 between twenty and forty, and 36 above forty. Three fourths of the cases are in males. In a few instances heredity has been adduced as a possible cause, and antecedent disease, such as syphilis, but this is doubtful. More important is local irritation, upon which Trousseau lays special stress, and gives instances in which chronic irritation of the skin, chronic nasal catarrh, or the irritation of a decayed tooth gave rise to local gland swellings, which preceded a general development of the disease. In a large majority of the cases the disease comes on insidiously, without any recognizable cause.

Morbid Anatomy.—*The Lymph Glands.*—In a few cases the enlarged glands are hard and firm, but in a majority the growth is soft and elastic. In the early stage the individual glands are isolated, not larger than almonds or walnuts, and readily separated and movable. When advanced the glands fuse together, and a group, as in the neck, may form a large tumor, the size of an orange or even of a cocoa-nut. About such masses the capsular tissues are hard and dense, forming a firm investment. A growth may perforate the capsule and invade contiguous parts, such as the muscles, skin, or the solid organs. On section, the tumor has a grayish-white appearance; it is smooth, and of variable consistence, either firm and dry or soft and juicy. Suppuration is most frequently seen when the growth reaches the skin. In the deep glands the formation of pus is rare. Caseation is not common; occasionally there are areas of necrosis very like it. The superficial glands are most often attacked, particularly the cervical groups, and the glands may be traced as continuous chains along the trachea and the carotids, uniting the axillary and mediastinal glands.

The axillary group is involved next in order of frequency, and the masses may pass beneath the pectorals and beneath the scapulæ. The inguinal glands occasionally form very large masses. Of the internal groups, those of the thorax are most often affected, either the chain in the posterior mediastinum or the bronchial group, or those of the anterior mediastinum. The trachea and the aorta with its branches may be completely surrounded by the growths, and be but little compressed. From the anterior mediastinum the masses may perforate the sternum and appear as an external tumor.

Of the abdominal groups, the retroperitoneal is most frequently involved and may form a continuous chain from the diaphragm to the inguinal canals, and extend into the pelvis. The glands may compress the ureters, involve the sacral or lumbar nerves, or compress the iliac veins. Occasionally they adhere to the uterus and broad ligament so as to simulate fibroids. I saw, some years ago, one of the most distinguished gynæcologists of Germany perform laparotomy in a case of this kind, in which the diagnosis of myomatous tumors of the uterus had been made. Occasionally the mesenteric or hepatic lymph glands may form large abdominal tumors.

Histologically the chief change is an increase in the cells, with or without thickening of the reticulum. In the early stage there is simple hyperplasia and the relations of the lymph paths are maintained, but when the glands are greatly enlarged the normal arrangement is disturbed. The reticulum varies extremely; in the softer growths it is expanded and can scarcely be found; in the harder structures the network of fibres is very distinct, and there is probably an increased development of the adenoid tissue.

Spleen.—In seventy-five per cent of the cases collected by Gowers this organ was hypertrophied, and in fifty-six of these cases it presented lymphoid growths. The enlargement is rarely great, and does not approximate to the large leukæmic spleen. The lymphoid tumors form grayish-white bodies ranging in size from a pea to a walnut, and may resemble lymph glands in appearance and consistence. Histologically, they consist of lymph corpuscles in a fibrous reticulum.

The marrow of the long bones may be converted into a rich lymphoid tissue; in a few instances the pyoid form, such as is more common in leukæmia, has been found. The tonsils may be involved and the follicles at the root of the tongue. Occasionally secondary growths are seen in the intestines.

The liver is often enlarged and may present scattered lymphoid tumors. The kidneys are occasionally involved and are the seat of growths similar to those of the spleen and liver. The lungs are occasionally directly attacked from the bronchial glands at the root, and secondary nodules may be found throughout their substance. Pleural effusions are not uncommon. Involvement of the nervous system is rare, but paraplegia may be induced by invasion of the spinal canal. The skin may be the seat of adenoid growths, as in a case reported by Greenfield.

Symptoms.—Enlargement of the glands of the neck, axilla, or groins is usually the first symptom noticed. In a few cases the anæmia and constitutional symptoms attract attention before the glandular involvement is evident. When the trouble begins in the deeper groups, pressure effects may be first noticed; thus, paroxysmal dyspnœa with pain in the chest may result from enlargement of the bronchial glands before any physical signs can be detected. Œdema of the feet and shooting

pains in the nerves were the first symptoms in one case which I dissected for Ross, and in another case at the Montreal General Hospital there was paraplegia from pressure on the cord. Such instances, however, are exceptional, and in the majority of cases the swelling of the superficial glands is the earliest symptom. Epistaxis has occasionally been noted, but not so frequently as in leukæmia. With progressive enlargement of the glands the patient becomes anæmic.

Usually, the cervical group is first affected, and it may be impossible to decide whether the enlargement is syphilitic, tuberculous, or lymphadenomatous. One side is first affected as a rule, and it may be months, or even, as in one of my cases, three years before the affection extends to other groups. Ultimately huge tumors may develop, which obliterate the neck and extend upon the shoulders and over the clavicles and sternum. The trachea is surrounded, great dyspnœa is produced, and not infrequently tracheotomy is necessary. In the later stages, the skin becomes involved and ulcerates. The axillary group may form large tumors, which compress the brachial or axillary veins and cause swelling of the arms. The inguinal glands are not so often involved, but may form large or even pendulous tumors.

In the thoracic glands, as mentioned, the various groups may be involved and produce pressure upon the veins or upon the trachea. In a case at present under observation the superior cava is completely obliterated and a very extensive collateral circulation has been established by means of the mammary and epigastric veins. The skin over the sternum is a mass of fluctuating veins, some of which contain phleboliths. In the abdomen the mesenteric glands may be enlarged, or more commonly the retroperitoneal group. When the patient is thin there may be no difficulty in detecting these, but in stout persons the diagnosis may be impossible. In connection with the affections of the abdominal glands there may be bronzing of the skin, which was well marked in Case IV of my series. A remarkable feature is the variations in the rate of growth and in the size of the glands. They may reduce rapidly and almost disappear from a region, and before death the tumors may diminish very much. The spleen may be enlarged and readily palpable. The thyroid also may be involved, and in a few instances the thymus has been affected. Though present in a majority of the cases, there may be enormous enlargement of the lymph glands without marked anæmia. In one of my cases the blood-corpuscles did not sink below 4,000,000 per cubic millimetre, and in only one instance have I counted the blood below 2,000,000. The red blood-corpuscles rarely show extreme poikilocytosis. The white corpuscles may be moderately increased and the lymphocytes most abundant. Occasionally the leucocytes are greatly increased and the characters of the blood become those of a lymphatic leukæmia. Nucleated red blood-corpuscles may be present, but not in such numbers as in leukæmia.

Of cardiac symptoms, palpitation is common. Hæmic murmurs are

often heard over the heart. Shortness of breath may be due to the anæmia, to pressure upon the trachea, or, in some instances, to pleuritic effusion associated with mediastinal growths. Fever is observed in nearly all cases; even in the early stages there is slight elevation. It may be of an irregular hectic type, or continuous, with evening exacerbation. Very remarkable are the cases with ague-like paroxysms, which may persist for weeks or months. They were present in Case I of my series. Pel, of Amsterdam, has given a thorough description of these attacks, and Ebstein has described a case under the remarkable title of "Chronic Recurrent Fever, a New Infectious Disease." In his case during nine months the attacks were present for periods of from twelve to fourteen days and alternated with apyrexia for ten or eleven days.

The digestive symptoms are usually not marked. It is not uncommon to find albumen in the urine. Headache, giddiness, and noises in the ear may be associated with the anæmia. Delirium and coma may be present. Deafness may be produced by growth of the adenoid tissue in the pharynx close to the Eustachian tubes. Inequality of the pupils may be present, owing to pressure of the glands on the cervical sympathetic. The skin may show definite secondary lymphatic tumors, bronzing may occur, and occasionally a most intense and troublesome prurigo.

Diagnosis.—A tuberculous adenitis may at first be very difficult to differentiate. The chief points of distinction are as follows: Tuberculous adenitis is more common in the young and involves the submaxillary group of glands more frequently than those of the anterior and posterior cervical triangles, which are usually affected first in Hodgkin's disease. The enlargement may last for years in a group without extending. The bunches are often, when small, welded together and, most important of all, tend to suppurate—a feature rarely seen in true lymphadenoma, except when it has attained very large size. Strict limitation to one side of the neck or to the axilla is suggestive of tuberculous disease rather than lymphadenoma.

There is an acute tuberculous adenitis, which may involve the lymph glands of the neck, producing enormous enlargement. A man, aged twenty-four, was admitted to the General Hospital, Montreal, with great swelling of the cervical glands on both sides, tonsillitis, and sloughing pharyngitis, with irregular fever and diarrhœa. The case was at first regarded as one of Hodgkin's disease. The occurrence of rigors and intermittent pyrexia is in favor of lymphadenoma. There are cases in which it may for a time be impossible to make a diagnosis. When the glands are only moderately enlarged on one side of the neck or axilla, they should be removed, and the diagnosis can then be thoroughly established.

Prognosis.—Recovery is very rare. The course of the disease is extremely variable. Early and rapid growth in the mediastinal groups may produce pressure effects and cause death before the development is extreme. In some cases the enlargements spread rapidly and group after

group becomes involved in a few months. These acute cases may run a course in three or four months. Chronic cases may last for three or four years. Periods of quiescence are not uncommon. The tumors may not only cease to grow, but gradually diminish and even disappear, without special treatment. Usually a cachexia develops, the anæmia progresses, and there are dropsical symptoms. The mode of death is usually by asthenia; less commonly by pressure from a tumor; and occasionally by coma.

Treatment.—When small and localized the glands should be removed. Local applications are of doubtful benefit. I have never seen special improvement follow the persistent use of iodine or the various ointments.

Arsenic has a positive value in the disease. It should be given in increasing doses, and stopped when unpleasant effects are manifested. The results have in many instances been striking. Due allowance must be made for the fluctuations in the size of the growths which occur spontaneously. I have seen no ill effects from the administration of Fowler's solution for months at a time, and many patients have taken from fifteen to twenty minims three times a day for weeks, and in some instances for months. Recoveries have been reported under this treatment. Personally, no instance of recovery has come under my notice in the cases of which I have notes. Phosphorus is recommended by Gowers and Broadbent, and should be used if the arsenic is not well borne. Quinine, iron, and cod-liver oil are useful as tonics. Every possible means must be taken to support the patient's strength.

IV. ADDISON'S DISEASE.

Definition.—A constitutional affection characterized by asthenia, depressed circulation, irritability of the stomach, and pigmentation of the skin. In a majority of the cases it is associated with tuberculous disease of the adrenals, in other instances with wasting of these organs or with changes in the abdominal sympathetic system.

The recognition of the disease is due to Addison, of Guy's Hospital, whose monograph on The Constitutional and Local Effects of Disease of the Suprarenal Capsules was published in 1855.

Etiology.—Males are more frequently attacked than females. In Greenhow's analysis of 183 cases 119 were males and 64 females. A majority of the cases occur between the twentieth and the fortieth year. A congenital case has been described in which the skin had a yellow-gray tint. The child lived for eight weeks, and post mortem the adrenals were found to be large and cystic. Injury, such as a blow upon the abdomen or back, and caries of the spine have in many cases preceded the attack. The disease is rare in America. Eight cases have come under my personal observation, either clinically or anatomically.

See admirable paper on Tuberculosis of the adrenals by Alezais et Armand Rev. de Med 1891

Morbid Anatomy and Pathology.—There is rarely emaciation or anæmia. In a great majority of the cases the adrenals are affected. There may be (*a*) atrophy of one or both glands, due to an interstitial cirrhosis, of which cases have been described by Hadden and Goodhart. (*b*) Tuberculosis, which is the common condition. The capsules are thickened and present firm caseous masses, surrounded by connective tissue. There is usually much fibrous thickening and matting of the adjacent structures, and the affection has definitely been shown to be tuberculous. Tuberculous lesions are common in other parts, particularly in the lungs, though in a number of the cases tuberculosis has been limited to the adrenals. (*c*) There may be malignant disease of the adrenals, which has been present in a few instances of genuine Addison's disease. Among other anatomical features the condition of the abdominal sympathetic has been specially studied. The nerve-cells of the semilunar ganglia have been described as degenerated and deeply pigmented, and the nerves sclerotic. The ganglia are not uncommonly entangled in the cicatricial tissue about the adrenals. The spleen has occasionally been found enlarged; the thymus may persist and be larger than normal.

It is difficult to explain satisfactorily all the symptoms of this remarkable disease. The theories which have been advanced are briefly as follows: (*a*) That the disease depended upon the loss of function of the adrenals. This was the view of Addison. It is held that the blood is gradually poisoned by the retention of some material, the destruction or alteration of which is a function of the suprarenals; (*b*) that it is an affection of the abdominal sympathetic system, induced most commonly by disease of the adrenals, but also by other chronic affections which involve the solar plexus and its ganglia. According to this view, it is an affection of the nervous system, and the pigmentation has its origin in changes induced through the trophic nerves. The pronounced debility is the outcome of disturbed tissue metabolism, and the circulatory, respiratory, and digestive symptoms are due to implication of the pneumogastric. The changes found in the abdominal sympathetic are held to support this view, and its advocates urge the occurrence of pigmentation of the skin in tuberculosis of the peritonæum, cancer of the pancreas, or aneurism of the abdominal aorta. Opposed to it are the facts that the lesions described in the sympathetic system are indefinite, and identical changes occur without the symptoms of Addison's disease.

Symptoms.—In the words of Addison the characteristic symptoms are "anæmia, general languor or debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin."

The pigmentation is the symptom which, as a rule, first attracts attention. The grades of coloration range from a light yellow to a deep brown, or even black. In typical cases it is diffuse, but always deeper on the exposed parts and in the regions where the normal pigmentation is

most intense. At first it may be confined to the face and hands. Occasionally it is absent. Patches of atrophy of pigment, leucoderma, may occur. The pigmentation is found on the mucous membranes of the mouth, conjunctivæ, and vagina. A patchy pigmentation of the serous membranes has often been found. The anæmia, upon which Addison laid stress, is of a moderate grade. It was not present in a marked degree in any of my cases.

Gastric disturbances are common; nausea and vomiting may be early and prominent symptoms; diarrhœa, too, is frequent, and may come on without cause. The pulse is small and rapid, and the heart's action feeble. Sometimes there is a special liability to syncope. One of the most pronounced features of the disease is the profound asthenia, which is out of all proportion to the general condition. The patient complains of a lack of energy, both mental and bodily; the least exertion is an effort, and may be followed by giddiness or noises in the ears. Headache is a frequent symptom. With the advancement of the disease the prostration becomes more marked, the patient remains in bed, the voice gets weak, the intelligence dulled, and death occurs either by syncope or gradual asthenia. Occasionally there are convulsions. The urine is usually normal. Polyuria has been described. The urinary pigments have been found increased.

Diagnosis.—Pigmentation of the skin is not confined to Addison's disease. The following are the conditions which may give rise to an increase in the pigment:

(1) Abdominal growths—tubercle, cancer, or lymphoma. In tuberculosis of the peritonæum pigmentation is not uncommon.

(2) Pregnancy, in which the discoloration is usually limited to the face, the so-called *masque des femmes enceinte.* Uterine disease is a common cause of a patchy melasma.

(3) Hepatic disease, which may induce definite pigmentation, as in the diabetic cirrhosis. More commonly in overworked persons of constipated habit and with sluggish livers there is a patchy staining about the face and forehead.

(4) The vagabond's discoloration, caused by the irritation of lice and dirt, which may reach a very high grade, and has sometimes been mistaken for Addison's disease.

(5) In rare instances there is deep discoloration of the skin in melanotic cancer, so deep and general that it has been confounded with *melasma suprarenale.*

(6) In certain cases of exophthalmic goitre abnormal pigmentation occurs, as noted by Drummond and others.

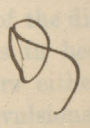
In any case of unusual pigmentation these various conditions must be sought for, and the diagnosis of Addison's disease is scarcely justifiable without the asthenia. In many instances it is difficult early in the disease to arrive at a definite conclusion. The occurrence of

An endemic pigment disease has been described by Gomez in the Cordilleras - in which deposits of pigment occur in large blotches on the face and limbs.

Hutchinson's Arch. of Surg. Vol. 5 - p. 134

The healing Art

~~Experiment & medicine~~ has made no more brilliant advance
than in ~~the treatment of~~ the cure of certain disorders due to disturbed function of the Thyroid Gland. That we can
today rescue children doomed to helplessness & idiocy, that we
can restore the unfortunate victims to life the hopeless victims of
myxoedema is a triumph ~~directly due to~~ of Experimental
Medicine



and the disease prevails endemically at the
Eastern end of Lake Ontario, about Kingston, and
in parts of the State of Michigan (Dock)

fainting fits, of nausea, and gastric irritability is an important indication.

Prognosis.—The disease is usually fatal. The cases in which the bronzing is slight or does not occur run a more rapid course. There are occasionally acute cases which, with great weakness, vomiting, and diarrhœa, prove fatal in a few weeks. In a few cases the disease is much prolonged, even to six or ten years. In rare instances recovery has taken place, and periods of improvement, lasting many months, may occur.

Treatment.—The causal indications cannot be met. When there is profound asthenia the patient should be confined to bed, as fatal syncope may at any time occur. In three of my cases death was sudden. When anæmia is present iron may be given in full doses. Arsenic and strychnia are useful tonics. For the diarrhœa large doses of bismuth should be given; for the irritability of the stomach, creosote, hydrocyanic acid, ice, and champagne. The diet should be light and nutritious. Many patients thrive best on a strictly milk diet.

V. DISEASES OF THE THYROID GLAND.

Goitre.

Definition.—Hypertrophy of the thyroid gland, occurring sporadically or endemically.

In this country sporadic cases are common. Endemically it is found particularly in the mountainous regions of Switzerland and in parts of Italy. No satisfactory explanation has been given of the existence of the disease in this form.

Anatomically the following varieties may be distinguished: (*a*) Parenchymatous, in which the enlargement is general and the follicles, usually newly formed, contain a gelatinous colloid material. (*b*) Vascular, in which the enlargement is chiefly due to dilatation of the blood-vessels without the new formation of glandular tissue. (*c*) Cystic goitre, in which the enlarged gland is occupied by large cysts, the walls of which often undergo calcification.

Symptoms.—The enlargement may be uniform throughout the entire gland, or affect only one lobe, or the isthmus alone. When small, a goitre causes no inconvenience. In its growth it may compress the trachea, causing dyspnœa, or may pass beneath the sternum and compress the veins. These, however, are exceptional circumstances, and in a large proportion of all cases no serious symptoms are noted. The affection usually comes under the care of the surgeon. Sudden death occasionally occurs in large bronchoceles. In some instances it may be difficult to determine the cause and it has been thought to be associated with pressure on the vagi. I have reported an instance in which it resulted from hæmor-

rhage into the gland and into the adjacent tissues. The blood passed into the cellular tissues of the neck and into the sternum, covering the aorta and pericardium.

Tumors of the Thyroid.

These are very varied. (*a*) Adenomata, either simple or malignant. The latter may form extensive metastases. A case is reported by Hayward in which growths resembling thyroid tissue occurred in the lungs and various bones of the body. (*b*) Cancer, of which several forms have been described. (*c*) Sarcoma. All of these have a surgical rather than a medical interest.

It may be mentioned that the aberrant or accessory thyroid gland may form large tumors in the mediastinum or in the pleura. I have reported two cases of this kind,* and an instance is on record in which an enormous cystic accessory thyroid occupied the entire right pleura.

Exophthalmic Goitre (*Graves's Disease; Basedow's Disease*).

Definition.—A disease of unknown origin, characterized by exophthalmos, enlargement of the thyroid, and functional disturbance of the vascular system.

Etiology.—The disease is rare in men. The age of onset is usually from the twentieth to the thirtieth year. It is sometimes seen in several members of the same family. Worry, fright, and depressing emotions precede the development of the disease in a number of cases.

Morbid Anatomy and Pathology.—No constant changes have been found in exophthalmic goitre. Special attention has been paid to the condition of the sympathetic system, as the rapid action of the heart and dilatation of the vessels has been attributed to paralysis of the sympathetic fibres, particularly the vaso-dilators. This view has found many supporters, but neither in the ganglia nor in the nerves are there any changes which can be regarded as constant and peculiar (Hale White). On the other hand, many features of the disease are explicable on the view that it is an affection of the medulla oblongata, and Hale White has reported a case dying of an acute intercurrent disease in which there were hæmorrhages in the floor of the fourth ventricle. The vascular and nervous features might be due to a lesion of this part; but it is difficult on any theory to explain all the symptoms of the disease and to bring into line the mental and vascular phenomena, the exophthalmos and the goitre.

Symptoms.—Acute and chronic forms may be recognized. In the acute form the disease may develop with great rapidity. In a patient of J. H. Lloyd's, of Philadelphia, a woman, aged thirty-nine, who had been considered perfectly healthy, but whose friends had noticed that for

* Medical News, 1890.

The medicinal treatment of goitre is very unsatisfactory. Iodine & various combinations & formally; Iodide of potash internally, ergot & many other drugs are recommended. The thyroid extract has been used successfully by Bruns in many of these cases.

+ Miss Herzberg - 1624 Head ave. . seen 4.20.92.
2 sisters in the family with exophthalmic goitre.
one aged 26 other 19.

Dr. Platt's brother who died under my care. Had Jan 1894 had a sister with the disease.

In the private ward - Jeff. Hosp (1.29.94) are two sisters one with the other, exophthalmic goitre & the other with exophthalmic goitre. a third sister had goitre & recovered.

see Tabs.

Strumitis, abscess inflammation of the thyroid

(

1 See Greenfield that it is a thyroid disease.

or more

which may be heard also over the manubrium. These vascular murmurs are very rare in simple goitre

some time her eyes looked rather prominent, was suddenly seized with intense vomiting and diarrhœa, rapid action of the heart, and great throbbing of the arteries. The eyes were prominent and staring and the thyroid gland was found much enlarged and soft. The gastro-intestinal symptoms continued, the pulse became more rapid, the vomiting was incessant, and the patient died on the third day of the illness; only the abdominal and thoracic organs could be examined and no changes were found. Two rapidly fatal cases occurred at the Philadelphia Hospital, one of which, under F. P. Henry's care, had marked cerebral symptoms. More frequently the onset is gradual and the disease is chronic. The three characteristic symptoms vary a good deal in their onset. Cardiac and vascular symptoms are usually first to develop and the patient complains of palpitation with breathlessness, and on examination the impulse is found to be increased in force, the apex beat is in normal position, the carotids throb, and the abdominal aorta pulsates visibly. This is one of the conditions in which the capillary pulse and the pulsation in the veins of the hands are occasionally seen. The pulse-rate at first may not be more than 95 or 100, but when the disease is established may reach 140 or 160. Any emotional excitement sets the heart beating with great intensity, and on exposure of the skin of the upper part of the chest a transient hyperæmia is seen. Soft murmurs are not uncommon at the base of the heart. In the long-standing cases the heart may be hypertrophied and the sounds very intense. In rare instances they may be heard some distance from the patient; according to Graves, as far as four feet.

Exophthalmos usually follows the vascular disturbance. It is readily recognized by the protrusion of the balls, and partly by the fact that the lids do not completely cover the sclerotics, so that a rim of white is seen above and below the cornea. The protrusion may become very great and the eye may even be dislocated from the socket. The vision is normal. Graefe noted that when the eyeball is moved downward the upper lid does not follow it as in health. This is known as Graefe's sign. The palpebral aperture is wider than in health, owing to spasm or retraction of the upper lid (Stellwag's sign). Changes in the pupils and in the optic nerves are rare. Pulsation of the retinal arteries is common.

The enlargement of the thyroid commonly develops with the exophthalmos. It may be general or in only one lobe, and is rarely as large as in ordinary goitre. The vessels are usually much dilated, and the whole gland may be seen to pulsate. A thrill may be felt on palpation and on auscultation a loud systolic murmur, or more commonly a *bruit de diable*. Among other symptoms which may develop are anæmia, emaciation, and slight fever. Attacks of vomiting and diarrhœa may occur. Tremor is present in many cases, involving the hands, and is usually very fine. The greatest complaint is of the unpleasant throbbing in the arteries, often accompanied with unpleasant flushes of heat and profuse perspirations. Skin symptoms are not infrequent—pigmentation, patches of leucoderma,

or atrophy of pigment, and urticaria. In the very acute case above referred to, urticaria was a prominent symptom. Irritability of temper, change in disposition, and great mental depresssion have been described. An important complication is acute mania, in which the patient may die in a few days. Symptoms of general paresis have been noted in a few cases. A feature of interest noted by Charcot is the great diminution in the electrical resistance, which may be due to the saturation of the skin with moisture owing to the vaso-motor dilatation (Hirt). Bryson has noted the fact that the chest expansion may be greatly diminished.

The course of the disease is usually chronic, lasting several years. After persisting for six months or a year the symptoms may disappear. There are remarkable instances in which the symptoms have come on with great intensity, following fright, and have disappeared again in a few days. A certain proportion of the cases recover, but when the disease is well developed recovery is rare.

Treatment.—Medicinal measures are notoriously uncertain. The combination of digitalis and iron may be tried, and, when there is anæmia, often does good. I have never seen any advantage from the use of aconite or veratrum viride. The tincture of strophanthus will sometimes reduce the rapidity of the heart's action. Ergot is warmly recommended by some writers. Belladona gives relief occasionally, and should be administered until the dryness of the throat is obtained. No measures are so successful as rest in bed with an ice-bag or Leiter's tube applied occasionally over the heart, or, what is sometimes more agreeable, over the lower part of the neck and manubrium sterni. I have known the pulse to be reduced in this way from 140 to 90. Electricity has been much lauded and instances of cure have been reported. In many cases temporary improvement certainly follows the use of the galvanic current, the cathode being placed at the back of the neck and the anode along the course of the sympathetic or over the heart. Treatment of the thyroid gland itself is rarely successful, and the operative measures have not been very satisfactory. Ligation of the arteries of the thyroid has been tried.

Myxœdema.

Definition.—A constitutional affection, characterized clinically by a myxœdematous condition of the subcutaneous tissues and mental failure, and anatomically by atrophy of the thyroid gland. The disease was described by Sir William Gull as a cretinoid change, and by Ord as a special disease, to which he gave the above name.

Clinical Forms.—Three groups of cases may be recognized: (*a*) *Congenital form*, or sporadic cretinism. In these cases there is congenital absence of the thyroid, and the child is a dwarf, having a thick neck, short arms and legs, and prominent abdomen. The face is large, the lips are

loss of hair

The relation of the symptoms to nasal obstruction is interesting. (see Putnam Oper)

Belladonna 60 drops

O'Donovan gave it for two years continuously the drops could be given a day without untoward symptoms but without curing the case.

Good reference to the literature in D. A. f. Nervenheilkunde. Bd 3 and 1893. Gottstein

Cretinism in America. Plutin cases.

Graves disease + Myxoedema occurred in sisters who were under my care.

Most frequently tuberculosis (Greenfield)

thick, the tongue is large and usually protrudes. The mental condition is that of imbecility or idiocy.

(*b*) *Myxœdema Proper.*—In this, women are very much more frequently affected than men—in a ratio of one to six. The disease may affect several members of a family, and it may be transmitted through the mother. In some instances there has been first the appearance of exophthalmic goitre. Though occurring most commonly in women, it seems to have no special relation to the catamenia or to pregnancy, though in one instance the symptoms of myxœdema disappeared during pregnancy. It is not so common in this country as in England. The symptoms of this form, as given by Ord,* are marked increase in the general bulk of the body, a firm, inelastic swelling of the skin, which does not pit on pressure, dryness and roughness, which tend, with the swelling, to obliterate in the face the lines of expression, imperfect nutrition of the hair, local tumefaction of the skin and subcutaneous tissues, particularly in the supraclavicular region. The physiognomy is altered in a remarkable way, the features are coarse and broad, the lips thick, the nostrils broad and thick, and the mouth is enlarged. Over the cheeks, sometimes the nose, there is a reddish patch. There is a striking slowness of thought and of movement. The memory becomes defective, the patients become irritable and suspicious, and there may be headache. In some instances there are delusions and hallucinations, leading to a final condition of dementia. The gait is heavy and slow. The temperature may be below normal. The functions of the heart, lungs, and abdominal organs are normal. Hæmorrhage sometimes occurs. Albuminuria is sometimes present, more rarely glycosuria. Death is usually due to some intercurrent disease. The thyroid gland is diminished in size and may become completely atrophied and converted into a fibrous mass. The subcutaneous fat is abundant and in one or two instances a great increase in the mucin has been found.

The course of the disease is slow but progressive, and extends over ten or fifteen years. I have recently had under observation a case to which the term acute myxœdema might be applied. A young man, aged twenty, presented a gradual enlargement of the face, particularly of the lips and cheeks and nose, without actual œdema. The backs of the hands were also swollen, but did not pit. The condition came on with enlargement of the thyroid, and, after persisting for between three and four months, is now gradually subsiding.

(*c*) *Operative Myxœdema; Cachexia Strumipriva.*—Horsley, in a series of interesting experiments, showed that complete removal of the thyroid in monkeys was followed by the production of a condition similar to that of myxœdema and often associated with spasms or tetanoid contractures, and followed by apathy and coma. When the monkeys were kept warm myxœdema was averted, and, instead of an acute myxœdema, the animals devel-

* Report on Myxœdema, Clinical Society's Transactions, 1888.

oped a condition which closely resembled cretinism. An identical condition may follow extirpation of the thyroid in man. Kocher, of Bern, found that after complete extirpation a cachectic condition followed in many cases, the symptoms of which are practically identical with those of myxœdema. The disease follows only a certain number of total and a much smaller proportion of partial removals of the thyroid gland. Of 408 cases, in 69 the operative myxœdema developed. It has been thought that if a small fragment of the thyroid remains, or if there are accessory glands, which in animals are very common, these symptoms do not develop. It is possible that in men, in the cases of complete removal, the accessory fragments subserve the function of the gland. Operative myxœdema is very rare in this country; the only case of which I know is a patient of McGraw's, of Detroit.

It is evident that the thyroid gland supplies some essential secretion of first importance to normal metabolism. What this is or how it acts is at present beyond our knowledge.

The *diagnosis* of the disease is easy. Bright's disease is the only condition for which it could be readily mistaken, but the absence of pitting, the curious condition of the face, and the absence of albumen in the urine are features which would readily distinguish it.

Unfortunately, no satisfactory treatment is known. The patients suffer in cold and improve greatly in warm weather. They should, therefore, be kept at an even temperature, and should, if possible, move to a warm climate during the winter months.

~~Whom was that to~~ ~~anyone~~ ~~who knows about~~

The general aspect of the patient - the subcutaneous swelling ~~yellow~~ and the ~~yellow~~ pallor - ~~suggest~~ Bright's disease which may be strengthened by the discovery of tube casts & albumen in the urine; but the ~~dried~~ character of the swelling, the ~~effect of gravity~~ the exceeding dryness of the skin, the yellowish white colour, the low temperature, the loss of hair, & the dull listless mental state should suffice to differentiate the two conditions.

which, he informs me has recovered under the use of the thyroid & heart

There are certain conditions which simulate or may even be associated with myxoedema (a) The supra-clavicular

c Sclerodermia.

(b) Dercum has described a muscular tissue dystrophy - adiposis dolorosa - in which irregular areas there - a development of subcutaneous masses of soft tissue associated with shooting pains & disturbances of sensation

SECTION VII.

DISEASES OF THE KIDNEYS.

I. ANOMALIES IN FORM AND POSITION.

Anomalies in Form.—These rarely come within the scope of the physician. Atrophy or congenital absence of one kidney is associated with great enlargement of the other organ. Fused kidneys may have a horse-shoe shape, or both organs may form a large mass, which is often displaced, being either in an iliac fossa or in the middle line of the abdomen, or even in the pelvis. Under these circumstances it may be mistaken for a new growth. In Polk's case the organ was removed under the belief that it was a floating kidney.* The patient lived eleven days, had complete anuria, and it was found post mortem that a single unsymmetrical kidney, as this form is called, had been removed.

Movable Kidney

(*Floating Kidney; Palpable Kidney; Ren mobilis; Nephroptosis*).

The kidney is held in position by its fatty capsule, by the peritonæum which passes in front of it, and by the blood-vessels. The lower edge of the left kidney is nearly two inches from the iliac crest, a little below the level of the second lumbar spine; that of the right is usually from one half to three quarters of an inch lower. Normally the kidney is firmly fixed, but under certain circumstances one or another organ, more rarely both, becomes movable. In rare cases the kidney is surrounded, to a greater or less extent, by the peritonæum, and is anchored at the hilus by a mesonephron. Some would limit the term floating kidney to this condition.

Movable kidney is almost always acquired. It is most common in women. Of the 667 cases collected in the literature by Kuttner, 584 were in women and only 83 in men. It is more common on the right than on the left side. Of 727 cases analyzed by this author, it occurred on the right in 553 cases, on the left in 81, and on both sides in 93. The

* New York Medical Journal, 1883.

greater frequency of the condition in women may be attributed to compression of the lower thoracic zone by tight lacing, and, more important still, to the relaxation of the abdominal walls which follows repeated pregnancies. This does not account for all the cases, as movable kidney is by no means uncommon in nulliparæ. Drummond believes that in a majority of the cases there is a congenitally relaxed condition of the peritoneal attachments. Wasting of the fat about the kidney may be a cause in some instances. Trauma and the lifting of heavy weights are occasionally factors in its production. The kidney is sometimes dragged down by tumors. The greater frequency on the right side is probably associated with the position of the kidney just beneath the liver, and the depression to which the organ is subjected with each descent of the diaphragm in inspiration.

And, lastly, movable kidney is met with in many cases which present that combination of neurasthenia with gastro-intestinal disturbance which has been described by Glénard * as *enteroptosis*.

To determine the presence of a movable kidney the patient should be placed in the dorsal position, with the head moderately low and the abdominal walls relaxed. The left hand is placed in the lumbar region behind the eleventh and twelfth ribs; the right hand in the hypochondriac region, in the nipple line, just under the edge of the liver. Bimanual palpation may detect the presence of a firm, rounded body just below the edge of the ribs. If nothing can be felt the patient should be asked to draw a deep breath, when, if the organ is palpable, it is touched by the fingers of the right hand. Various grades of mobility may be recognized. It may be possible barely to feel the lower edge on deep palpation—*palpable kidney*—or the organ may be so far displaced that on drawing the deepest breath the fingers of the right hand may be in a thin person slipped above the upper end of the organ, which can be readily held down, but cannot be pushed below the level of the navel—*movable kidney*. In a third group of cases the organ is freely movable, and may even be felt just above Poupart's ligament, or may be in the middle line of the abdomen, or can even be pushed over beyond this point. To this the term *floating kidney* is appropriate, whether the organ has a mesonephron or not.

And, lastly, a dislocated kidney may become fixed in an abnormal position. This is extremely rare, and in a very large number of cases I have found only one instance of the kind. A woman, aged twenty-nine, with four children, had nervous symptoms with abdominal pain, and had been much worried by the discovery of a tumor, just to the right of the middle line, close to the navel. It was not movable, but the distinctly reniform shape and the depression at the left margin indicated that it was doubtless a dislocated kidney which had become fixed.

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* Revue de Médécine, 1887; Pourcelot, Paris Thesis, 1889.

Position of head - sometimes better high - or a little to raised.

The movable kidney is not painful on pressure, except when it is grasped very firmly, when there is a dull pain, or sometimes a sickening sensation. Examination of the patient from behind may show a distinct flattening in the lumbar region on the side in which the kidney is mobile.

Symptoms.—In a large majority of cases the condition gives no trouble, and it is well, if detected accidentally, not to let the patient know of its presence. In other instances there is pain in the lumbar region or a sense of dragging and discomfort, or there may be intercostal neuralgia. In a large group the symptoms are those of neurasthenia with dyspeptic disturbance. In women the hysterical symptoms may be marked, and in men various grades of hypochondriasis. The gastric disturbance is usually a form of nervous dyspepsia. Dilatation of the stomach has been observed, owing, as suggested by Bartels, to pressure of the dislocated kidney upon the duodenum. This view has been supported by Oser, Landau, and Ewald. On the other hand, Litten holds that the dilatation of the stomach is the cause of the mobility of the kidney, and he found in 40 cases of depression and dilatation of the stomach 22 instances of dislocation of the kidney on the right side. My own experience coincides with that of Drummond, who has very exceptionally found the two conditions to coexist. While not denying the possibility of causal relationship between the two, it seems probable, considering the frequency of floating kidney, that the complication is only a coincidence. The association, however, with a *depressed* stomach is certainly not uncommon in women. Constipation is not infrequent. Some writers have described pressure upon the gall-ducts, with jaundice, but it is not very likely to occur.

Under the name *enteroptosis*, Glénard has described a special symptom group characterized by nervous dyspepsia, prolapse of the abdominal organs, particularly the transverse colon, with looseness of the mesenteric and peritoneal attachments, so that there is a falling down of the viscera (splanchnoptosis). Dilatation of the stomach and mobility of the kidney are very commonly associated with this state. Glénard held that he could feel the prolapsed transverse colon as a narrow band, but Ewald states correctly that this is the pancreas, which in many of these cases can be distinctly palpated. According to Glénard, the kink in the colon causes the constipation, while the depression of the stomach and intestines leads to vascular disturbance and impairment of the motor and secretory functions.

In floating kidney there are attacks (simulating gastralgia or renal colic) characterized by severe abdominal pain, chills, nausea, vomiting, fever, and collapse. Scarcely any mention is made of such symptoms, which were first described by Dietl in 1864, and a more wide-spread knowledge of their occurrence in connection with this condition is desirable. My attention was called to them in 1880 by Palmer Howard in the case of a stout lady, who suffered repeatedly with the most severe attacks of abdominal pain and vomiting, which constantly required morphia. A

tumor was discovered a little to the right of the navel, and the diagnosis of probable neoplasm was concurred in by Flint (Sr.) and Gaillard Thomas. The patient lost weight rapidly, became emaciated, and in the spring of 1881 again went to New York, where she saw Van Buren, who diagnosed a floating kidney and said that these paroxysms were associated with it in a gouty person. He cut off all stimulants, reassured the lady that she had no cancer, and from that time she rapidly recovered, and the attacks have been few and far between. In this patient any overindulgence in eating or in drinking is still liable to be followed by a very severe attack. These attacks may also be mistaken for renal colic, and the operation of nephrotomy has been performed.

In other instances the attacks of pain may be thought to be due to intestinal disease or to recurring appendicitis. The cause of these paroxysmal attacks is not quite clear. Dietl thought they were due to strangulation of the kidney or to twists or kinks in the renal vessels due to the extreme mobility. During the attacks the urine is sometimes high-colored and contains an excess of uric acid or of the oxalates. It is stated, too, that blood or pus may be present. The kidney may be tender, swollen, and less freely movable. Intermittent hydronephrosis has sometimes been associated with movable kidney.

The *diagnosis* is rarely doubtful, as the shape of the organ is usually distinctive and the mobility marked. Tumors of the gall-bladder, ovarian growths, and tumors of the bowels may in rare instances be confounded with it.

Treatment.—The kidney has been extirpated in many instances, but the operation is not without risk, and there have been several fatal cases. Stitching of the kidney—nephrorrhaphy—as recommended by Hahn, is the most suitable procedure, and statistics recently published by Keen show that relief is afforded in many cases by the procedure. It does not, however, always succeed.

The treatment by trusses and bandages is not satisfactory, though great relief is sometimes obtained. As a rule, bandages, with pads pressing to the right of the navel, are not well borne, as the kidney is often sensitive. In some instances, however, the greatest relief is experienced by this procedure. An air-pad beneath the bandage, as recommended by Newman, is probably the best. In other cases a broad bandage well padded in the lower abdominal zone pushes up the intestines and makes them act as a support. In the attacks of severe colic morphia is required. When dependent, as seems sometimes the case, upon an excess of uric acid or the oxalates, the diet must be carefully regulated.

II. CIRCULATORY DISTURBANCES.

Normally the secretion of urine is accomplished by the maintenance of a certain blood-pressure within the glomeruli and by the activity of the renal epithelium. Bowman's views on this question have been generally accepted, and the watery elements are held to be filtered from the glomeruli; the amount depending on the rapidity and the pressure of the blood current; the quality, whether normal or abnormal, depending upon the integrity of the capillary and glomerular epithelium; while the greater portion of the solid ingredients are excreted by the epithelium of the convoluted tubules. The integrity of the epithelium covering the capillary tufts within Bowman's capsule is essential to the production of a normal urine. If under any circumstances their nutrition fails, as when, for example, the rapidity of the blood-current is lowered, so that they are deprived of the necessary amount of oxygen, the material which filters through is no longer normal (i. e., water), but contains serum albumen. Cohnheim has shown that the renal epithelium is extremely sensitive to circulatory changes, and that compression of the renal artery for only a few minutes causes serious disturbance.

The circulation of the kidney is remarkably influenced by reflex stimuli coming from the skin. Exposure to cold causes heightened blood-pressure within the kidneys and increased secretion of urine. So also in the chills of malaria, after which a large amount of pale urine may be passed.

Congestion of the Kidneys.—(1) *Active Congestion; Hyperæmia.*—Acute congestion of the kidney is met with in the early stage of nephritis, whether due to cold or to the action of poisons and severe irritants. Turpentine, cubebs, cantharides, and copaiba are all stated to cause extreme hyperæmia of the organ. The most typical congestion of the kidney which we see post mortem is that in the early stage of acute Bright's disease, when the organ may be large, soft, of a dark color, and on section blood drips from it freely.

It has been held that in all the acute fevers the kidneys are congested, and that this explained the scanty, high-colored, and often albuminous urine. On the other hand, by Roy's oncometer, Walter Mendelson has shown that the kidney in acute fever is in a state of extreme anæmia, small, pale, and bloodless; and that this anæmia, increasing with the pyrexia and interfering with the nutrition of the glomerular epithelium, accounts for the scanty, dark-colored urine of fever and for the presence of albumen. In the prolonged fevers, however, it is probable that relaxation of the arteries again takes place. Certainly it is rare to find post mortem such a condition of the kidney as is described by Mendelson. On the contrary, the kidney of fever is commonly swollen, the blood-vessels are congested, and the cortex frequently shows traces of cloudy swelling. However, the circulatory disturbances in acute fevers are probably less im-

portant than the irritative effects of either the specific agents of the disease or the products produced in their growth, or in the altered metabolism of the tissues. The urine is diminished in amount, and may contain albumen and tube-casts.

(2) *Passive Congestion; Mechanical Hyperæmia.*—This is found in cases of chronic disease of the heart or lung, with impeded circulation, and as a result of pressure upon the renal veins by tumors, the pregnant uterus, or ascitic fluid. In the cardiac kidney, as it is called, the cyanotic induration associated with chronic heart-disease, the organs are enlarged and firm, the capsule strips off, as a rule, readily, the cortex is of a deep red color, and the pyramids of a purple red. The section is coarse-looking, the substance is very firm, and resists cutting and tearing. The interstitial tissue is increased, and there is a small celled infiltration between the tubules. Here and there the Malpighian tufts have become sclerosed. The blood-vessels are usually thickened, and there may be more or less granular, fatty, or hyaline changes in the epithelium of the tubules. The condition is indeed a diffuse nephritis. The urine is usually reduced, is of high specific gravity, and contains more or less albumen. Hyaline tube-casts and blood-corpuscles are not uncommon. In uncomplicated cases of the cyanotic induration uræmia is rare. On the other hand, in the cardiac cases with extensive arterio-sclerosis, the kidneys are more involved and the renal function is likely to be disturbed.

III. ANOMALIES OF THE URINARY SECRETION.

1. Hæmaturia.

The following division may be made of the causes of hæmaturia:

(1) *General Diseases.*—The malignant forms of the acute specific fevers, such as small-pox, malaria, yellow fever, etc.; scurvy, purpura, and hæmophilia. Occasionally in leukæmia hæmaturia occurs.

(2) *Renal Causes.*—Acute congestion and inflammation, as in Bright's disease, or the effect of toxic agents, such as turpentine, carbolic acid, and cantharides. When the carbolic spray was in use many surgeons suffered from hæmaturia in consequence of this poison. Renal infarction, as in ulcerative endocarditis. New growths, in which the bleeding is usually profuse. Tubercle rarely causes hæmaturia, though at the onset, when the papillæ are involved, there may be bleeding. Stone in the kidney is a frequent cause. Parasites: The *filaria sanguinis hominis* and the *Bilharzia* cause a form of hæmaturia met with in the tropics. The echinococcus is rarely associated with hæmorrhage.

(3) *Affections of the Urinary Passages.*—Stone in the ureter, malignant disease or ulceration of the bladder, the presence of a calculus, parasites, and, very rarely, ruptured veins in the bladder. Bleeding from the

I v Anuria —

Total suppression of urine occurs under the following conditions

(1) as a rare event in the ~~acute~~ vascular changes of acute nephritis. ~~For a time~~ No urine may be formed for a time

2 Much more commonly complete anuria is seen in the paralysis of ~~renal~~ shock when frequent blood both urea ~~often~~ ~~more or less pain~~. There may be no discomfort and careful search ~~is~~ made in vain for ~~the~~ six or eight days have passed before the function was restored. Cabot reports cases of recovery after suppression had existed for nearly eight days In a calculus subject ~~the indications are to the surgeon~~ ~~and~~ should be sought for ~~to be~~ acted & explore the ureters thoroughly from in front

3

urethra occasionally occurs in gonorrhœa and as a result of the lodgment of a calculus.

(4) *Traumatism.*—Injuries may produce bleeding from any part of the urinary passages. By a fall or blow on the back the kidney may be ruptured, and this may be followed by very free bleeding; less commonly the blood comes from injury of the bladder or of the prostate. Blood from the urethra is frequently due to injury by the passage of a catheter, or sometimes to falls or blows.

And, lastly, there are cases in which hæmaturia occurs for a long time without discoverable cause, particularly in young persons. The health may not be seriously impaired. Gull has characterized, in a happy way, a case of this kind as one of renal epistaxis.

Of special interest is the malarial hæmaturia which prevails in certain districts and has already been considered in the section on paludism.

The *diagnosis* of hæmaturia is usually easy. The color of the urine varies from a light smoky to a bright red, or it may have a dark porter color. Examined with the microscope, the blood-corpuscles are readily recognized, either plainly visible and retaining their color, in which case they are usually crenated, or simply as shadows. In ammoniacal urine or urines of low specific gravity the hæmoglobin is rapidly dissolved from the corpuscles, but in normal urine they remain for many hours unchanged.

Other tests are rarely necessary. The guaiacum test consists of the addition to the urine, in a test-tube, of a drop or two of the tincture of guaiacum and two minims of ozonic ether. A blue color forms at the line of contact of the two fluids and diffuses itself through the ether.

The spectroscopical examination of the urine may show either the single band of reduced hæmoglobin or the double band of oxyhæmoglobin between the lines D and E.

It is important to distinguish between blood coming from the bladder and from the kidneys, though this is not always easy. From the bladder the blood may be found only with the last portions of urine, or only at the termination of micturition. In hæmorrhage from the kidneys, the blood and urine are intimately mixed. Clots are more commonly found in the blood from the kidneys, and may form moulds of the pelvis or of the ureter. When the seat of the bleeding is in the bladder, on washing out this organ, the water is more or less blood-tinged; but if the source of the bleeding is higher, the water comes away clear. In many instances it is difficult to settle the question by the examination of the urine alone, and the symptoms and the physical signs must also be taken into account.

2. Hæmoglobinuria.

This condition is characterized by the presence of blood-pigment in the urine. The blood-cells are either absent or in insignificant numbers. The coloring matter is not hæmatin, as indicated by the old name, *hæma-*

tinuria, nor in reality always hæmoglobin, but it is most frequently methæmoglobin. The urine has a red or brownish-red, sometimes quite black color, and usually deposits a very heavy brownish sediment. When the hæmoglobin occurs only in small quantities, it may give a lake or smoky color to the urine. Microscopical examination shows the presence of granular pigment, sometimes fragments of blood-disks, epithelium, and very often darkly pigmented urates. The urine is also albuminous. The number of red blood-corpuscles bears no proportion whatever to the intensity of the color of the urine. Examined spectroscopically, there are either the two absorption bands of oxyhæmoglobin, which is rare, or, more commonly, there are the three absorption bands of methæmoglobin, of which the one in the red near *C* is characteristic. Two clinical groups may be distinguished.

(1) **Toxic Hæmoglobinuria.**—This is caused by poisons which produce rapid dissolution of the blood-corpuscles, such as chlorate of potash in large doses, pyrogallic acid, carbolic acid, arseniuretted hydrogen, carbon dioxide, naphthol, and muscarine; also the poisons of scarlet fever, yellow fever, typhoid fever, malaria, and syphilis. It has also followed severe burns. Exposure to excessive cold and violent muscular exertion are stated to produce hæmoglobinuria. A most remarkable toxic form occurs in horses, coming on with great suddenness and associated with paresis of the hind legs. Death may occur in a few hours or a few days. Horses are attacked only after being stalled for some days and then taken out and driven, particularly in cold weather. The affection is common in horses in this country. The form of hæmoglobinuria from cold and exertion is extremely rare No instance of it, even in association with frost-bites, came under my observation in Canada. Blood transfused from one mammal into another causes dissolution of the corpuscles with the production of hæmoglobinuria; and, lastly, there is the *epidemic hæmoglobinuria* of the new-born, associated with jaundice, cyanosis, and nervous symptoms.

(2) **Paroxysmal Hæmoglobinuria.**—This rare disease is characterized by the occasional passage of bloody urine, in which the coloring matter only is present. It is more frequent in males than in females, and occurs chiefly in adults. It seems specially associated with cold and exertion, and has often been brought on, in a susceptible person, by the use of a cold foot-bath. Paroxysmal hæmoglobinuria has been found, too, in persons subject to the various forms of Raynaud's disease. Many regard the relation between these two affections as extremely close; some hold that they are manifestations of one and the same disorder. Druitt, the author of the well-known Surgical Vade-mecum, has given a graphic description of his sufferings, which lasted for many years, and were accompanied with local asphyxia and local syncope. The connection, however, is not very common. In only one of the cases of Raynaud's disease which I have seen was paroxysmal hæmoglobinuria present, and in it epileptic attacks occurred at the same time. The relation of the disease to

Weir's disease.

and Thunderclaps disease.

malaria is not so close as has been thought by many writers. No doubt it has been frequently confounded with a malarial hæmaturia. The attacks may come on suddenly after exposure to cold or as a result of mental or bodily exhaustion. They may be preceded by chills and pyrexia. In other instances the temperature is subnormal. There may be vomiting and diarrhœa. Pain in the lumbar region is not uncommon. The hæmoglobinuria rarely persists for more than a day or two—sometimes, indeed, not for a day. There are instances in which, even in the course of a single day, there have been two or three paroxysms, and in the intervals clear urine has been passed. Jaundice has been present in a number of cases. According to Ralfe, paroxysmal hæmoglobinuria may alternate with general symptoms of the same character, but associated only with the passage of albumen and an increased quantity of urea in the urine. In such cases he supposes that the toxic agent, whatever its nature, has destroyed only a limited number of the corpuscles, the coloring matter of which is readily dealt with by the spleen and liver, while the globulin is excreted in the urine. The cases are rarely if ever fatal.

The essential pathology of the disease is unknown, and it is difficult to form a theory which will meet all the facts—particularly the relation with Raynaud's disease, which is rightly regarded as a vaso-motor disorder. Increased hæmolysis and dissolution of the hæmoglobin in the blood-serum (hæmoglobinæmia) precedes, in each instance, the appearance of the coloring matter in the urine; but, as Ponfick has shown, the amount of free hæmoglobin must reach a certain grade before it is excreted.

Treatment.—In all forms of hæmaturia rest is essential. In that produced by renal calculi the recumbent posture may suffice to check the bleeding. Full doses of acetate of lead and opium should be tried, then ergot, gallic and tannic acid, and the dilute sulphuric acid. The oil of turpentine, which is sometimes recommended, is a risky remedy in hæmaturia. Extr. hamamelis virgin. and extr. hydrastis canad. are also recommended. Cold may be applied to the loins or dry cups in the lumbar region.

The treatment of hæmoglobinuria is unsatisfactory. Nothing seems to check the occurrence of the attacks. During the paroxysm the patient should be kept warm and given hot drinks. Quinine is recommended in large doses, on the supposition—as yet unwarranted—that the disease is specially connected with malaria. If there is a syphilitic history iodide of potassium, in full doses, may be tried. In a warm climate the attacks are much less frequent.

III. Albuminuria.

The presence of albumen in the urine, formerly regarded as indicative of Bright's disease, is now recognized as occurring under many circumstances without the existence of serious organic change in the kidney.

Two groups of cases may be recognized—those in which the kidneys show no coarse lesions, and those in which there are evident anatomical changes.

Albuminuria without Coarse Renal Lesions.—(*a*) *Functional, So-called Physiological, Albuminuria.*—In a normal condition of the kidney only the water and the salts are allowed to pass from the blood. When albuminous substances transude there is probably disturbance in the nutrition of the epithelium of the capillaries of the tuft, or of the cells surrounding the glomerulus. This statement is still, however, in dispute, and Senator, Grainger Stewart, and others hold that there is a physiological albuminuria which may follow muscular work, the ingestion of food rich in albumen, violent emotions, cold bathing, and dyspepsia. The differences of opinion on this point are striking, and observers of equal thoroughness and reliability have arrived at directly opposite conclusions. The presence of albumen in the urine, in any form and under any circumstance, may be regarded as indicative of change in the renal or glomerular epithelium, a change, however, which may be transient, slight, and unimportant, depending upon variations in the circulation or upon the irritating effects of substances taken with the food or temporarily present, as in febrile states.

Much attention has been given of late years to the albuminuria of adolescence, or *cyclic albuminuria*, which is also believed to be a functional disorder. A majority of the cases occur in young persons—boys more commonly than girls—and the condition is often discovered accidentally. The urine, as a rule, contains only a very small quantity of albumen, but in some instances large quantities are present. The most striking feature is the variability. It may be absent in the morning and only present after exertion, or it may be greatly increased after taking food, particularly proteids. The quantity of urine may be but little if at all increased, the specific gravity is usually normal, and the color may be high. Occasionally, hyaline casts may be found, and in some instances there has been transient glycosuria. As a rule, the pulse is not of high tension and the second aortic sound is not accentuated.

Various forms of this affection have been recognized by writers, such as neurotic, dietetic, cyclic, intermittent, and paroxysmal—names which indicate the characters of the different varieties. A large proportion of the cases get well after the condition has persisted for a variable period. This in itself is an evidence that the changes, whatever their nature, were transient and slight. In these instances the albumen exists in small quantity, tube-casts are not present, and the arterial tension is not increased. In a second group the albumen is more persistent, the amount is larger, though it may vary from day to day, and the pulse tension is increased. In such instances the persistent albuminuria probably indicates actual organic change in the kidney.

(*b*) *Febrile Albuminuria.*—Pyrexia, by whatever cause produced,

may cause slight albuminuria. The presence of the albumen is due to slight changes in the glomeruli induced by the fever, such as cloudy swelling, which cannot be regarded as an organic lesion. It is extremely common, occurring in pneumonia, diphtheria, typhoid fever, and even in the fever of acute tonsillitis. The amount of albumen is slight, and it usually disappears from the urine with the cessation of the fever.

(*c*) *Hæmic Changes.*—Purpura, scurvy, chronic poisoning by lead or mercury, syphilis, leukæmia, and profound anæmia may be associated with slight albuminuria. Abnormal ingredients in the blood, such as bile-pigment and sugar, may cause the passage of small amounts of albumen.

The transient albuminuria of pregnancy may belong to this hæmic group, although in a majority of such cases there are changes in the renal tissue. Albumen may be found sometimes after the inhalation of ether or chloroform.

(*d*) Albuminuria occurs in certain affections of the *nervous system.* This so-called neurotic albuminuria is seen after an epileptic seizure and in apoplexy, tetanus, exophthalmic goitre, and injuries of the head.

Albuminuria with Definite Lesions of the Urinary Organs.—(*a*) Congestion of the kidney, either active, such as follows exposure to cold and is associated with the early stages of nephritis, or passive, due to obstructed outflow in disease of the heart or lungs, or to pressure on the renal veins by the pregnant uterus or tumors.

(*b*) Organic disease of the kidneys—acute and chronic Bright's disease, amyloid and fatty degeneration, suppurative nephritis, and tumors.

(*c*) Affections of the pelvis, ureters, and bladder, when associated with the formation of pus.

Tests for Albumen.—Both morning and evening urine should be examined, and in doubtful cases at least three specimens. If turbid, the urine should be filtered, though turbidity from the urates is of no moment, since it disappears at once on the application of heat.

Heat and Nitric-acid Test.—The urine is boiled in a test-tube over a spirit-lamp, and a drop of nitric acid is then added. If a cloudiness occurs on boiling, it may be due to phosphates, which are dissolved on the addition of an acid. Persistence of the cloudiness indicates albumen.

Heller's Test.—A small quantity of fuming nitric acid is poured into the test-tube, and with a pipette the urine is allowed to flow gently down the side upon the acid. At the line of junction of the two fluids, if albumen is present, a white ring is formed. This contact method is trustworthy, and, for the routine clinical work, is probably the most satisfactory. A diffused haze, due to mucin, is sometimes seen just above the white ring of albumen. A colored ring at the junction of the acid and the urine is due to the oxidation of the coloring matters in the urine.

Sir William Roberts strongly recommends the *magnesium-nitric test.* One volume of strong nitric acid is mixed with five volumes of the saturated

solution of sulphate of magnesium. This is used in the same way as the nitric acid in Heller's test.

Picric acid, introduced by George Johnson, is a delicate and useful test for albumen. A saturated solution is used and employed as in the contact method. It has been urged against this test that it throws down the mucin, peptones, and certain vegetable alkaloids, but these are dissolved by heat.

For minute traces of albumen the trichloracetic acid may be used, or Millard's fluid, which is extremely delicate and consists of glacial carbolic acid (ninety-five per cent), 2 drachms; pure acetic acid, 7 drachms; liquor potassæ, 2 ounces 6 drachms.

A quantitative estimate of the albumen can be made by means of Esbach's tube, but the rough method of heating and boiling a certain quantity of acidulated urine in a test-tube and allowing it to stand, is often employed. The depth of deposit can then be compared with the whole amount of urine, and the proportion is expressed as a mere trace, almost solid, one fourth, one half, and so on. This, of course, does not give an accurate indication of the proportion of albumen in the total quantity of urine. For the more elaborate methods the reader is referred to the works on urinalysis.

The above tests refer entirely to serum albumen. Other albuminous substances occur, such as serum globulin, peptones, and hemialbumose. By saturating the urine with magnesium sulphate, the globulin is precipitated, coagulated, and then readily separated from the serum albumen.

Traces of *peptones* are found in the urine in many acute diseases and in chronic suppuration. They are not precipitated by heat or nitric acid, but are thrown down by picric acid and dissolved by heat. If the urine contains peptones, a rose or pinkish tint is formed at the junction of the two fluids when urine is allowed to flow gently into a test-tube containing Fehling's solution. Peptonuria has no clinical significance.

Propepton, or hemialbumose, is not of any practical importance. It was found by Bence-Jones in the urine in osteomalacia, and occurs occasionally in other affections.

Prognosis.—This depends, of course, entirely upon the cause. Febrile albuminuria is transient, and in a majority of the cases depending upon hæmic causes the condition disappears and leaves the kidneys intact. An occasional trace of albumen in a man over forty, with or without a few hyaline casts, and with increased tension and thick vessel walls, usually indicates changes in the kidneys. The persistence of a slight amount of albumen in young men without increased arterial tension is less serious as even after continuing for years it may disappear. I have already spoken of the outlook in the so-called cyclic albuminuria.

Practically in all cases the presence of albumen indicates a change of some sort in the glomeruli, the nature, extent, and gravity of which it is difficult to estimate, so that other considerations, such as the presence of

tube-casts, the existence of increased tension, the general condition of the patient, and the influence of digestion upon the albumen, must be carefully considered.

The physician is daily consulted as to the relation of albuminuria and life assurance. As his function is to protect the interests of the company, he should reject all cases in which albumen occurs in the urine. It is even doubtful if an exception should be made in young persons with transient albuminuria. Naturally, companies lay great stress upon the presence or absence of albumen, but in the most serious and fatal malady with which they have to deal, chronic interstitial nephritis, the albumen is often absent or transient, even when the disease is well developed. After the fortieth year, from a standpoint of life insurance, the state of the arteries is far more important than the condition of the urine.

IV. Pyuria (*Pus in the Urine*).

Causes.—(1) *Pyelitis and Pyelonephritis.*—In large abscesses of the kidney, pyonephrosis, the pus may be intermittent, and for days or even weeks the urine is free. In calculous and tuberculous pyelitis the pyuria is usually continuous, though varying in intensity. In these cases, as a rule, the pus is mixed with the urine, which is acid in reaction. In the early stages of pyelitis the transitional epithelium may be abundant, but is not in any way distinctive. In the pyelitis and pyelonephritis following cystitis the urine is usually alkaline, and contains more mucus; micturition is usually more frequent, and the history points to a previous bladder affection.

(2) *Cystitis.*—The urine is alkaline, often fetid, the pus ropy, and the amount of urine greatly increased. The ropy, thick mucus usually comes with the last portions of the urine. Triple phosphate crystals may be present in the freshly passed urine.

(3) *Urethritis*, particularly gonorrhœa. The pus appears first, is in small quantities, and there are signs of local inflammation.

(4) In *leucorrhœa* the quantity of pus is usually small, and large flakes of vaginal epithelium are numerous. In doubtful cases, when leucorrhœa is present, the urine should be withdrawn by a catheter.

(5) *Rupture of Abscesses into the Urinary Passages.*—In such cases as pelvic or perityphlitic abscess there have been previous symptoms of pus formation. A large amount is passed within a short time, then the discharge stops abruptly or rapidly diminishes within a few days.

Pus gives to the urine a white or yellowish-white appearance. On settling there is a heavy grayish sediment, and the supernatant fluid is usually turbid. The sediment is often tenacious and ropy. The reaction is generally alkaline, and the odor may be ammoniacal even when passed. Examination with the microscope reveals the presence of a large number of pus-corpuscles, which are usually, when the pus comes from the blad-

der, well formed; the protoplasm is granular, and often shows many translucent processes.

The only sediment likely to be confounded with pus is that of the phosphates; but it is whiter and less dense, and is distinguished immediately by microscopical examination.

With the pus there is always more or less epithelium from the bladder and pelvis, but since in these situations the forms of cells are practically identical, they afford no information as to the locality from which the pus has come.

The treatment of pus in the urine is considered under the conditions in which it occurs.

V. Chyluria—Non-parasitic.

This is a rare affection, occurring in temperate regions and unassociated with the *filaria hominis sanguinis*. The urine is of an opaque white color; it resembles milk closely, is occasionally mixed with blood (hæmatochyluria), and sometimes coagulates into a firm, jelly-like mass. In other instances there is at the bottom of the vessel a loose clot which may be distinctly blood-tinged. Under the microscope the turbidity seems to be caused by numerous minute granules—more rarely oil droplets similar to those of milk. Traces of albumen are usually present. The amount of urine passed is generally increased, and the chylous condition is intermittent. It may persist for years without deterioration of health or evidence of serious disease.

Since the discovery of the *filaria hominis sanguinis* it has been incorrectly held by some that all of the cases of chyluria are of this parasitic nature. I had an opportunity in Montreal of making a careful study of a French-Canadian woman, a patient of J. B. McConnell's, who had had chyluria for more than thirteen years. The urine was quite milky in color and occasionally mixed with blood. Neither ova nor embryos were found in the urine or in the blood examined at night. After her death I was enabled to make a thorough dissection of the abdominal lymph vessels, which were found perfectly normal. The thoracic duct was not enlarged, the renal lymphatics were not distended; the kidneys were increased in size, but showed no special changes. The most careful examination of the lymph glands and vessels failed to reveal the presence of parasites.

The pathology of the condition is unknown. No known remedies have any influence upon the chyluria.

(For parasitic chyluria see Filariasis.)

VI. Lithuria (*Lithæmia; Lithic-acid Diathesis*).

The amount of uric acid excreted daily depends greatly upon the diet, ranging from half a gramme on a vegetable to as high, even, as two

grammes on an animal diet. In the urine of herbivora it occurs only in traces. In that of carnivora it may be absent altogether. On the other hand, in the urine of birds and reptiles it is the chief nitrogenous ingredient. As Sir William Roberts remarks, its presence in the human urine is somewhat of an anomaly, as its place is very much better taken by urea, which is easily soluble and better adapted to the mammalian plan of a liquid urine. He regards it as a sort of vestigial remnant.

Place and Mode of Formation of the Uric Acid.—It is now very generally conceded that uric acid is formed in the tissues and excreted by the kidneys. It may occur in traces in the blood even in health. Von Jaksch, who has recently examined the blood of 109 individuals, found no trace in 9 healthy persons, nor was it present in cases of typhoid fever or in nervous affections or in diseases of the liver and gastro-intestinal canal, except when anæmia coexisted. On the other hand, it was present in connection with all those diseased processes in which oxidation was disturbed, either directly, as in affections of the lungs, such as pneumonia, or indirectly, as in anæmia, in which the oxygen-carriers are deficient. According to Haig, the amount in the blood rises and falls with the degree of alkalinity (as more is held in solution), and all circumstances which increase this are associated with an increase in the amount of uric acid.

As to the place of formation, the experimental evidence points strongly to the liver, and, according to Minkowski, it is formed there by the synthesis of ammonia and lactic acid. The views, however, as to its place of production and the antecedents are by no means harmonious. Garrod still holds that the kidneys are concerned not only with its excretion, but with its formation. On the other hand, Ebstein thinks that it is chiefly produced in the muscles and in the bone marrow. Nor is it yet settled whether uric acid is only an intermediate step in the formation of urea or whether it has an independent origin.

Mode of Elimination.—Uric acid is extremely insoluble, a gramme requiring for its solution, at ordinary temperature, fourteen litres of water, and about half that amount at body temperature. In the 1,500 to 2,000 c. c. of urine passed in the day the uric acid could not be dissolved, but it is eliminated in combination as soluble salts, chiefly as urates of ammonium and sodium. The power in the blood of holding the uric acid in solution depends upon the degree of alkalinity; thus it has been long known that the excretion of uric acid some hours after breakfast is high. This is in what Sir William Roberts calls "the alkaline tide." Haig has shown that this excretion can be increased or diminished by increasing or diminishing the alkalinity of the blood; thus, under salicylate of soda, given in fifteen grain doses three times a day, the excretion of the uric acid is increased on the first and second days, and subsequently falls to the normal amount. He explains this by supposing that the salicylate finds a considerable quantity of uric acid stored in the liver, spleen, and other tissues, gets this into solution, and the greater part of it is passed in the urine. His obser-

vations indicate that alkalies, such as the phosphate of soda and compounds of salicylic acid, increase markedly the excretion of this ingredient in the urine, and also increase the amount of it in the blood, withdrawing it from the spleen and liver. On the other hand, acids, lead, and iron interfere with the solubility of the uric acid and with its elimination. A fact of great practical importance which he mentions is that lithia, "though a beautiful solvent of uric acid in a test-tube, yet when given to the human subject by mouth never reaches the uric acid at all, because it at once forms an insoluble compound with the phosphate of soda in the blood, thus removing from that fluid one of the natural solvents of uric acid, and diminishing its power of holding uric acid in solution." This is directly opposed to the prevalent ideas of the value of the lithia compounds in the uric-acid diathesis.

The pathology of uric acid is more a matter of defective elimination than of excessive formation. In conditions of the system associated with persistent diminished alkalinity of the blood the uric acid accumulates in the liver, spleen, and the joints. The degree of alkalinity of the kidney structure possibly, as Haig suggests, may have an influence in determining how much shall be excreted and how much retained, and, according to his views, it is this small remnant or overflow which accumulates in the blood and produces headache, high tension of the pulse, and mental depression, and when deposited in the joints causes gouty arthritis and the uratic lesions.

Occurrence in the Urine.—The uric acid occurs in combination chiefly with ammonium and sodium, forming the acid urates. In smaller quantities are the potassium, calcium, and lithium salts. The uric acid may be separated from its bases and crystallizes in rhombs or prisms, which are usually of a deep red color, owing to the staining of the urinary pigments. The sediment formed is granular and the groups of crystals look like grains of Cayenne pepper. It is very important not to mistake a deposit of uric acid for an excess. The deposition of numerous grains in the urine within a few hours after passing is more likely to be due to conditions which diminish the solvent power than to increase in the quantity. Of the conditions which cause precipitation of the uric acid Roberts gives the following: "(1) High acidity; (2) poverty in mineral salts; (3) low pigmentation; and (4) high percentage of uric acid." The grade of acidity is probably the most important element.

More common is the precipitation of amorphous urates, forming the so-called brick-dust or lateritious deposit, which has a pinkish color, due to the presence of urinary pigment. It is composed chiefly of the acid sodium urates. It occurs particularly in very acid urine of a high specific gravity. As the urates are more soluble in warm solutions they frequently deposit as the urine cools. Here, too, the deposition does not necessarily, indeed usually does not, mean an excessive excretion, but the existence of conditions favoring the deposit.

On the relation of gout and gravel are
views correctly put by Sir Wm Roberts."

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Murchison introduced the term lithæmia to designate certain symptoms due, as he supposed, to functional disturbance of the liver. Not only have his views been widely adopted, but, as is so often the case when we give the rein to theoretical conceptions of disease, the so-called manifestations of this state have so multiplied that some authors attribute to this cause a considerable proportion of the ailments affecting the various systems of the body. Thus one writer enumerates not fewer then thirty-nine separate morbid conditions associated with lithæmia. From what has been said as to our knowledge, or rather our lack of knowledge, of the mode of formation and elimination of uric acid it is very evident that the physiology of the subject must be widely extended before we are in a position to draw safe conclusions. Thus it is by no means sure that, as Murchison supposed, the essential defect is in a functional disorder of the liver, disturbing the metabolism of the albuminous ingredients, nor is it at all certain that the only offending substance is uric acid. Bouchard contends that the so-called lithiasis has little or nothing to do with disturbance in the function of the liver, and that it has not been shown that uric acid is the only or even the chief agent in producing the symptoms. In the present imperfect state of knowledge it is impossible with any clearness to define the pathology of the so-called uric-acid diathesis. We may say that certain symptoms arise in connection with defective food or tissue metabolism, more particularly of the nitrogenous elements. Deficient oxidation is probably the most essential factor in the process, with the result of the formation of less readily soluble and less readily eliminated products of retrograde metamorphosis. This faulty metabolism if long continued may lead to gout, with uratic deposits in the joints, acute inflammations, and arterial and renal disease. In a large group of cases the disturbed metabolism produces high tension in the arteries (probably as a direct sequence of interference with the capillary circulation) and ultimately degenerations in various tissues, particularly the scleroses.

Overeating and overdrinking, when combined with deficient muscular exercise, lie at the basis of this nutritional disturbance. The symptoms which are believed to characterize the uric-acid diathesis have already been briefly considered under the section on irregular gout, and the question of diet and exercise has also been there considered.

VII. Oxaluria.

Oxalic acid occurs in the urine, in combination with lime, forming an oxalate which is held in solution by the acid phosphate of soda. About .01 to .02 gramme is excreted in the day. It never forms a heavy deposit, but the crystals—usually octahedra, rarely dumb-bell-shaped—collect in the mucus-cloud and on the sides of the vessel. The amount varies extremely with the diet, and it is increased largely when such fruits and vegetables as tomatoes and rhubarb are taken. It is also a product of incomplete oxi-

dation of the organic substances in the body, and in conditions of increased metabolism the amount in the urine becomes larger. It is stated also to result from the acid fermentation of the mucus in the urinary passages and the crystals are usually abundant in spermatorrhœa.

When in excess and present for any considerable time, the condition is known as oxaluria, the chief interest of which is in the fact that the crystals may be deposited before the urine is voided, and form a calculus. It is held by many that there is a special diathesis associated with this state and manifested clinically by dyspepsia, particularly the nervous form, irritability, depression of spirits, lassitude, and sometimes marked hypochondriasis. There may be in addition neuralgic pains and the general symptoms of neurasthenia. The local and general symptoms are probably dependent upon some disturbance of metabolism of which the oxaluria is one of the manifestations. It is a feature also in many gouty persons, and in the condition called lithæmia.

VIII. Cystinuria.

Cystin does not occur in normal urine. It is very rarely met with, and its chief interest is owing to the fact that it may form a calculus. Its presence in the urine has been determined in many members of the same family, and the condition appears sometimes to be hereditary. As it contains sulphur, it is thought to be formed from the taurin of the bile.

IX. Phosphaturia.

The phosphoric acid is excreted from the body in combination with potassium, sodium, calcium, and magnesium, forming two classes, the alkaline phosphates of sodium and potassium and the earthy phosphates of lime and magnesia. "*The alkaline phosphates* exist in the blood in the form of neutral sodium and potassium phosphates (hydrogen-disodium phosphates, HNa_2PO_4), but appear in the urine as acid sodium and potassium phosphates (dihydrogen-sodium phosphates, H_2NaPO_4), and thus cause the acid reaction of that secretion. The change of the neutral into the acid salt is caused by the decomposition effected by the act of secretion, in which the bicarbonates and neutral phosphates in the blood change into carbonates and acid phosphates respectively." (Ralfe.)

Of the *earthy phosphates*, those of lime are abundant, of magnesium scanty. In urine which has undergone the ammoniacal fermentation, either inside or outside the body, there is in addition the ammonio-magnesium or triple phosphate, which occurs in triangular prisms or in feathery or stellate crystals, hence the term given to this form of stellar phosphates. The earthy phosphates occur as a sediment in the urine when the alkalinity is due to a fixed alkali, or under certain circumstances the deposit may take place within the bladder, and then the phosphates are passed at the end of micturition as a whitish fluid, which is popularly

confounded with spermatorrhœa. The calcium phosphate may be precipitated by heat and produce a cloudiness which may be mistaken for albumen, but is at once dissolved upon making the urine acid. This condition is very frequent in persons suffering from dyspepsia or from debility of any kind. The phosphates may be in great excess, rising in the twenty-four hours to from 7 to 9 grammes (Tessier), whereas the normal amount is not more than 2·5 grammes. And, lastly, the phosphates may be deposited in urine which has undergone decomposition, in which the carbonate of ammonia from the urea combines with the magnesium phosphates, forming the triple salt. This is seen in cystitis, and is due to the introduction of a bacterial ferment.

The clinical significance of an excess of phosphates, to which the term phosphaturia is applied, has been much discussed. It must be remembered that a deposit does not necessarily mean an excess, to determine which a careful analysis of the twenty-four hours' secretion should be made. It has long been thought that there is a relation between the activity of the nerve-tissues and the output of phosphoric acid; but the question cannot yet be considered settled. The amount is increased in wasting diseases, such as phthisis, acute yellow atrophy of the liver, leukæmia, and severe anæmia, whereas it is diminished in acute diseases and during pregnancy.

In a condition termed by Tessier, Ralfe, and others phosphatic diabetes there is polyuria, thirst, emaciation, and a great increase in the excretion of phosphates, which may be as much as from seven to nine grammes in the day. The urine is usually acid, free from sugar, the patients are nervous; in some instances sugar has been present in the urine, and in others it subsequently makes its appearance.

X. Indicanuria.

The substance in the urine which has received this name is the indoxyl-sulphate of potassium, in which form it appears in the urine and is colorless. When concentrated acids or strong oxidizing agents are added to the urine, this substance is decomposed and the indigo set free. It is present only in small quantities in healthy urine. It is derived from the indol, a product formed in the small intestine by the decomposition of the albumen under the influence of bacteria. When absorbed, this is oxidized in the tissues to indoxyl, which combines with the potassium sulphate, forming the above-named substance.

The quantity of indican is increased on a milk diet, in all wasting diseases, and whenever any large quantities of albuminous substances are undergoing rapid decomposition, as in the severer forms of peritonitis and empyema. It is met with also in prolonged constipation and in ileus. Indican has occasionally been found in calculi. Though, as a rule, the urine is colorless when passed, there are instances in which the decompo-

sition has taken place within the body, and a blue color has been noticed immediately after the urine was voided. Sometimes, too, in alkaline urine on exposure there is a bluish film on the surface.

To test for indican, place four or five c. c. of nitric or hydrochloric acid in a test-tube; boil, and add an equal quantity of urine. A bluish ring develops at the point of contact. Add ten c. c. of chloroform and shake the test-tube, and on separation the chloroform has a violet or bluish color due to the presence of indican.

XI. Melanuria.

In melanotic cancer the urine, either at the time of voiding or after exposure to the air, may present a dark color. This pigment is known as melanin, and it may occur in solution or in the form of small granules. The urine may be voided clear, and subsequently, on exposure to the air or on the addition of oxidizing substances, becomes dark. In these cases it contains a chromogen called melanogen which turns dark by oxidation. Von Jaksch has found that "in urine containing melanin or its precursor, melanogen, Prussian blue is formed by adding a nitroprusside, aqueous potash, and an acid. This reaction, however, does not seem to depend on the presence of melanin, as it is not given by that substance when separated from the urine, but apparently by some other at present unknown substance, which is present in traces in normal urine and is increased in cases of melanuria, and also in those conditions where excess of indigo occurs in the urine." (Halliburton.)

XII. Other Substances.

Fat in the urine, or *lipuria*, occurs, according to Halliburton, first, without disease of the kidneys, as in excess of fat in the food, after the administration of cod-liver oil, in fat embolism occurring after fractures, in the fatty degeneration in phosphorus poisoning, in prolonged suppuration as in phthisis and pyæmia, in the lipæmia of diabetes mellitus; secondly, with disease of the kidneys, as in the fatty stage of chronic Bright's disease, in which fat casts are sometimes present, and, according to Ebstein, in pyonephrosis; and, thirdly, in the affection known as chyluria. The urine is usually turbid, but there may be fat drops as well, and fatty crystals have been found.

Lipaciduria is a term applied by von Jaksch to the condition in which there are volatile fatty acids in the urine, such as acetic, butyric, formic, and propionic.

Acetonuria.—Von Jaksch distinguishes the following forms of pathological acetonuria: The febrile, the diabetic, the acetonuria with certain forms of cancer, the form associated with inanition, acetonuria in psychoses, and the acetonuria which results from auto-intoxication. It is doubtful, however, whether the symptoms in these are really due to the

acetone. It may be the substances from which this is formed, particularly the diacetic acid or the oxybutyric acid. The odor of the acetone may be marked in the breath and evident in the urine. Le Nobel's test has been given in the section on diabetes.

Diacetic acid is probably never present in the urine in health. With a solution of ferric chloride it gives a Burgundy-red color. A similar reaction is given by acetic, formic, oxybutyric acids, and it may be present in the urine of patients who are taking antipyrin, thallin, and the salicylates. "If, however, the urine is previously boiled, diacetic acid, if present, still gives the ferric-chloride reaction, but these other substances do not. Fleischer found that the substance which gives the ferric-chloride reaction in diabetic urine is not taken up by ether after the urine has been acidulated with sulphuric acid, whereas ethyl-diacetic acid is soluble in ether." (Halliburton.)

Alcaptonuria.—Aromatic compounds occur in the urine after the administration of carbolic acid or gallic acid, and on exposure to air becomes dark. In carboluria the substance causing the black color is known as hydrochinon. Many years ago Boedeker met with cases in which the urine became dark, owing to the presence of an aromatic compound which he called alcapton. It has been found in cases of consumption, and in other instances in which there are no local lesions or no general disease. The urine may be clear on passing, and then darken on exposure to the air, or on the addition of liquor potassæ. The substance is apparently without clinical significance except in so far as it, with the other aromatic substances, is capable of reducing the Fehling solution, and may be mistaken for sugar.

Choluria and glycosuria have already been considered under jaundice and diabetes.*

IV. URÆMIA.

Under this term is grouped a series of manifestations, chiefly nervous, developing in the course of Bright's disease, and due to the retention within the blood of poisonous materials which should be eliminated in the urine.

Uræmia is usually seen in nephritis, but may occur when the ureters are obstructed, or when the circulation of blood in the kidneys is impeded, as in conditions of extreme engorgement following compression of the renal vessels or in the profound alterations of the blood in cholera.

Two opposite views are held with reference to the production of uræmia: (*a*) That it is due to the accumulation in the blood of excrementitious material—body poisons—which should be thrown off by the

* For further details concerning the urine the student is referred to von Jaksch's Clinical Diagnosis, Tyson on the Urine, and to Halliburton's Text-Book of Chemical Physiology and Pathology.

kidneys. "If, however, from any cause, these organs make default, or if there be any prolonged obstruction to the outflow of urine, accumulation of some or of all the poisons takes place, and the characteristic symptoms are manifested, but the accumulation may be very slow and the earlier symptoms, corresponding to the comparatively small dose of poison, may be very slight; yet they are in kind, though not in degree, as indicative of uræmia as are the more alarming, which appear towards the end, and to which alone the name uræmia is often given." (Carter.) Several poisons having distinct actions have been separated from the urine by Bouchard, two of which produce convulsions, and one of which is narcotic. Bouchard's observations tend strongly to confirm the view now generally held, that the symptoms are caused by the retention of the excretory products. The nature of these poisonous ingredients is not yet known. It was formerly thought that the urea was the offending substance, and it has been found increased in the blood in uræmia. Others hold that it is the accumulation of carbonate of ammonia. It is more probable, however, that there are several toxic agents at work.

(*b*) Traube suggested that the chief symptoms of uræmia, particularly the coma and convulsions, were due to localized œdema of the brain. In favor of this view is the fact that obstruction of the ureters, as by stone, does not necessarily produce uræmia, even if long continued, and in this obstructive suppression neither convulsions nor coma occur. Then, too, uræmia may supervene in a case of chronic Bright's disease in which a large amount of urine is being passed with a fair proportion of solids. Œdema of the brain certainly does occur in some fatal cases—it may be diffuse or localized, but it is not a constant lesion, and cannot explain all the symptoms of uræmia.

Symptoms.—Clinically, acute and chronic uræmia may be recognized, but, for convenience of description, it is perhaps best to follow the division of French writers into *cerebral*, *dyspnœic*, and *gastro-intestinal* forms.

Among the *cerebral* manifestations of uræmia may be described:

(*a*) *Mania.*—This may come on abruptly in an individual who has shown no previous indications of mental trouble, and who may not be known to have Bright's disease. In a remarkable case of this kind which came under my observation the patient became suddenly maniacal and died in six days. More commonly the delirium is less violent, but the patient is noisy, talkative, restless, and sleepless.

(*b*) *Delusional Insanity* (*Folie Brightique*).—Cases are by no means uncommon, and excellent clinical reports have been issued on the subject from several of the asylums of this country, particularly by Bremer, Christian, and Alice Bennett. Delusions of persecution are common. One of my cases committed suicide by jumping out of a window. The condition is of interest medico-legally because of its bearing on testamentary capacity. Profound melancholia may also supervene.

Sudden insensibility may occur after prolonged muscular exertion — as after running a ten-mile race. Cases have been reported in which unconsciousness came on rapidly with stertorous breathing and dilated pupils. Cases have occurred under conditions in which sun-stroke could be excluded — and Poore who reports a case in the Lancet (1879) considers that the condition is due to the too rapid accumulation of waste products in the blood — and hyperpyrexia from suspension of sweating.

An erythema may be present. The skin may be frosted with

(*c*) *Convulsions.*—These may come on unexpectedly or be preceded by pain in the head and restlessness. The attacks may be general and identical with those of ordinary epilepsy, though the initial cry may not be present. The fits may recur rapidly, and in the interval the patient is usually unconscious. Sometimes the temperature is elevated, but more frequently it is depressed, and may sink rapidly after the attack. Local or Jacksonian epilepsy may occur in most characteristic form in uræmia. A remarkable sequence of the convulsions is blindness—*uræmic amaurosis*—which may persist for several days. This, however, may occur apart from the convulsions. It usually passes off in a day or two. There are no ophthalmoscopic changes. Sometimes uræmic deafness supervenes, and is probably also a cerebral manifestation. It may also occur in connection with persistent headache, nausea, and other gastric symptoms.

(*d*) *Coma.*—Unconsciousness invariably accompanies the general convulsions, but a coma may develop gradually without any convulsive seizures. Frequently it is preceded by headache, and the patient gradually becomes dull and apathetic. In these cases there may have been no previous indications of renal disease, and unless the urine is examined the nature of the case may be overlooked. Twitchings of the muscles occur, particularly in the face and hands, but there are many cases of coma in which the muscles are not involved. In some of these cases a condition of torpor persists for weeks or even months. The tongue is usually furred and the breath very foul and heavy.

(*e*) *Local Palsies.*—In the course of chronic Bright's disease hemiplegia or monoplegia may come on spontaneously or follow a convulsion, and post mortem no gross lesions of the brain be found, but only a localized or diffused œdema. These cases, which are not very uncommon, may simulate almost every form of organic paralysis of cerebral origin.

(*f*) Of other cerebral symptoms, headache is important. It is most often occipital and extends to the neck. It may be an early feature and associated with giddiness. Other nervous symptoms of uræmia are intense itching of the skin, numbness and tingling in the fingers, and cramps in the muscles of the calves, particularly at night.

Uræmic dyspnœa is classified by Palmer Howard as follows: (1) Continuous dyspnœa; (2) paroxysmal dyspnœa; (3) both types alternating; and (4) Cheyne-Stokes breathing. The attacks of dyspnœa are most commonly nocturnal; the patient may sit up, gasp for breath, and evince as much distress as in true asthma. Occasionally the breathing is noisy and stridulous. The Cheyne-Stokes type may persist for weeks, and is not necessarily associated with coma. I have seen it in a man who travelled over a hundred miles to consult a physician. In another instance a patient, up and about, could only when at meals feed himself in the apnœa period. Though usually of serious omen and occurring with coma and

other symptoms, recovery may follow even after persistence for weeks or even months.

The *gastro-intestinal* manifestations of uræmia often set in with abruptness. Uncontrollable vomiting may come on and its cause be quite unrecognizable. A young married woman was admitted to my wards in the Montreal General Hospital with persistent vomiting of four or five days' duration. The urine was slightly albuminous, but she had none of the usual signs of uræmia, and the case was not regarded as one of Bright's disease. The vomiting persisted and caused death. The post-mortem showed extensive sclerosis of both kidneys. The attacks may be preceded by nausea and may be associated with diarrhœa. In some instances the diarrhœa may come on without the vomiting; sometimes it is profuse and associated with an intense catarrhal or even diphtheritic inflammation of the colon.

A special uræmic stomatitis has been described (Barie) in which the mucosa of the lips, gums, and tongue is swollen and erythematous. The saliva may be increased, and there is difficulty in swallowing and in mastication. The tongue is usually very foul and the breath heavy and fetid.

Diagnosis.—Uræmia may be confounded with:

(*a*) Cerebral lesions, such as hæmorrhage, meningitis, or even tumor. In apoplexy, which is so commonly associated with kidney disease and stiff arteries, the sudden loss of consciousness, particularly if with convulsions, may simulate a uræmic attack; but the mode of onset, the existence of complete hemiplegia, with conjugate deviation of the eyes, suggest hæmorrhage. As already noted, there are cases of uræmic hemiplegia or monoplegia which cannot be separated from those of organic lesion and which post mortem show no trace of coarse disease of the brain. I know of an instance in which a consultation was held upon the propriety of operation in a case of hemiplegia believed to be due to subdural hæmorrhage which post mortem was shown to be uræmic. Indeed, in some of these cases it is quite impossible to distinguish between the two conditions. So, too, cases of meningitis, in a condition of deep coma, with perhaps slight fever, furred tongue, and without localizing symptoms, may readily be confounded with uræmia.

(*b*) With certain infectious diseases. Uræmia may persist for weeks or months and the patient lies in a condition of torpor or even unconsciousness, with a heavily coated, perhaps dry, tongue, muscular twitchings, a rapid feeble pulse, with slight fever. This state not unnaturally suggests the existence of one of the infectious diseases. Cases of the kind are not uncommon, and I have known them to be mistaken for typhoid fever and for miliary tuberculosis.

(*c*) Uræmic coma may be confounded with poisoning by alcohol or opium. In opium poisoning the pupils are contracted; in alcoholism they are more commonly dilated. In uræmia they are not constant; they may be either widely dilated or of medium size. The examination of the eye-

Skin. post fuska.

ground should be made to determine the presence or absence of albuminuric retinitis. The urine should be drawn off and examined. The odor of the breath sometimes gives an important hint.

The condition of the heart and arteries should also be taken into account. Sudden uræmic coma is more common in the chronic interstitial nephritis. The character of the delirium in alcoholism is sometimes important, and the coma is not so deep as in uræmia or opium poisoning. It may for a time be impossible to determine whether the condition is due to uræmia, profound alcoholism, or hæmorrhage into the pons Varolii. The treatment will be considered under chronic Bright's disease.

V. ACUTE BRIGHT'S DISEASE.

Definition.—Acute diffuse nephritis, due to the action of cold or of toxic agents upon the kidneys.

In all instances changes exist in the epithelial, vascular, and intertubular tissues, which vary in intensity in different forms; hence writers have described a tubular, a glomerular, and an acute interstitial nephritis. Delafield recognizes *acute exudative* and *acute productive* forms, the latter characterized by proliferation of the connective-tissue stroma and of the cells of the Malpighian tufts.

Etiology.—The following are the principal causes of acute nephritis:

(1) Cold. Exposure to cold and wet is one of the most common causes. It is particularly prone to follow exposure after a drinking-bout.

(2) The poisons of the specific fevers, particularly scarlet fever, less commonly typhoid fever, measles, diphtheria, small-pox, chicken-pox, cholera, yellow fever, meningitis, and, very rarely, dysentery. Acute nephritis is not often associated with syphilis. In acute tuberculosis nephritis is not uncommon. It may also occur in septicæmia.

(3) Toxic agents, such as turpentine, cantharides, chlorate of potash, and carbolic acid may cause an acute congestion which sometimes terminates in nephritis. Alcohol probably never excites an acute nephritis.

(4) Pregnancy, in which the condition is thought by some to result from compression of the renal veins, although this is not yet finally settled. The condition may in reality be due to toxic products as yet undetermined.

(5) Acute nephritis occurs occasionally in connection with extensive lesions of the skin, as in burns or in chronic skin-diseases.

Morbid Anatomy.—The kidneys may present to the naked eye in mild cases no evident alterations. When seen early in more severe forms the organs are congested, swollen, dark, and the section may drip blood. In other instances the surface is pale and mottled, the capsule strips off readily, and the cortex is swollen, turbid, and of a grayish-red color, while the pyramids have an intense beefy-red tint. The glomeruli in some in-

stances stand out plainly, deeply swollen and congested; in other instances they are pale.

The histology may be thus summarized: (*a*) Glomerular changes. In a majority of the cases of nephritis due to toxic agents, which reach the kidney through the blood-vessels, the tufts suffer first, and there is either an acute intracapillary glomerulitis, in which the capillaries become filled with cells and thrombi, or involvement of the epithelium of the tuft and of Bowman's capsule, the cavity of which contains leucocytes and red blood-corpuscles. Hyaline degeneration of the contents and of the walls of the capillaries of the tufts is an extremely common event. These processes are perhaps best marked in scarlatinal nephritis. There may be proliferation about Bowman's capsule. These changes interfere with the circulation in the tufts and seriously influence the nutrition of the tubular structures beyond them.

(*b*) The alterations in the tubular epithelium consist in cloudy swelling, fatty change, and hyaline degeneration. In the convoluted tubules, the accumulation of altered cells with leucocytes and blood-corpuscles causes the enlargement and swelling of the organ. The epithelial cells lose their striation, the nuclei are obscured, and hyaline droplets often accumulate in them.

(*c*) Interstitial changes. In the milder forms a simple inflammatory exudate—serum mixed with leucocytes and red blood-corpuscles—exists between the tubules. In severer cases areas of small-celled infiltration occur about the capsules and between the convoluted tubes. These changes may be wide-spread and uniform throughout the organs or more intense in certain regions.

Symptoms.—The onset is usually sudden, and when the nephritis follows cold, dropsy may be noticed within twenty-four hours. After fevers the onset is less abrupt, but the patient gradually becomes pale and a puffiness of the face or swelling of the ankles is first noticed. In children there may at the outset be convulsions. Chilliness or rigors initiate the attack in a limited number of cases. Pain in the back, nausea, and vomiting may be present. The fever is variable. Many cases in adults have no rise in temperature. In young children with nephritis from cold or scarlet fever the temperature may, for a few days, range from 101° to 103°.

The most characteristic symptoms are the urinary changes. There may at first be suppression, more commonly the urine is scanty and highly colored and contains blood, albumen, and tube-casts. The quantity is reduced and only four or five ounces may be passed in the twenty-four hours; the specific gravity is high—1·025, or even more; the color varies from a smoky to a deep porter color, but is seldom bright red. On standing there is a heavy deposit; microscopically there are blood-corpuscles, epithelium from the urinary passages, and casts, hyaline, blood, and epithelial in character. The albumen is abundant, forming a curdy, thick pre-

cipitate. The total excretion of urea is reduced, though the percentage is high.

Anæmia is an early and marked symptom. In cases of extensive dropsy, effusion may take place into the pleuræ and peritoænum. There are cases of scarlatinal nephritis in which the dropsy of the extremities is trivial and effusion into the pleuræ extensive. The lungs may become œdematous. In rare cases there is œdema of the glottis. Epistaxis may occur or cutaneous ecchymoses may develop in the course of the disease.

The pulse may be hard, the tension increased, and the second aortic sound accentuated. Occasionally dilatation of the heart comes on rapidly and may cause sudden death (Goodhart). The skin is dry and it may be difficult to induce sweating.

Uræmic symptoms develop in a limited number of cases. They may occur at the onset with suppression, more commonly later in the disease. Ocular changes are not so common in acute as in chronic Bright's disease, but hæmorrhagic retinitis may occur and occasionally papillitis.

The course of acute Bright's disease varies considerably. The description just given is of the form which most commonly follows cold or scarlet fever. In many of the febrile cases dropsy is not a prominent symptom, and the diagnosis rests rather with the examination of the urine. Moreover, the condition may be transient and less serious. In other cases, as in the acute nephritis of typhoid fever, there may be hæmaturia and pronounced signs of interference with the renal function. The most intense acute nephritis may exist without anasarca.

In scarlatinal nephritis, in which the glomeruli are most seriously affected, suppression of the urine may be an early symptom, the dropsy is apt to be extreme, and uræmic manifestations are common. Acute Bright's disease in children, however, may set in very insidiously and be associated with transient or slight œdema, and the symptoms may point rather to affection of the digestive system or to brain-disease.

Diagnosis.—It is very important to bear in mind that the most serious involvement of the kidneys may be manifested only by slight œdema of the feet or puffiness of the eyelids, without impairment of the general health. The first indication of trouble may be a uræmic convulsion. This is particularly the case in the acute nephritis of pregnancy, and it is a good rule for the practitioner, when engaged to attend a case, invariably to ask that during the seventh and eighth months the urine should occasionally be sent for examination.

In nephritis from cold and in scarlet fever the symptoms are usually marked and the diagnosis is rarely in doubt. As already mentioned, every case in which albumen is present must not be called acute Bright's disease, not even if tube-casts be present. Thus the common febrile albuminuria, although it represents the first link in the chain of events leading to acute Bright's disease, should not be placed in the same category.

There are occasional cases of acute Bright's disease with anasarca, in

which albumen is either absent or present only as a trace. This is a rare condition. Tube-casts are usually found, and the absence of albumen is rarely permanent. The urine may be reduced in amount.

The character of the casts is of use in the diagnosis of the form of Bright's disease, but scarcely of such extreme value as has been stated. Thus, the hyaline and granular casts are common to all varieties. The blood and epithelial casts, particularly those made up of leucocytes, are most common in the acute cases.

Prognosis.—The outlook varies somewhat with the cause of the disease. Recoveries in the form following exposure to cold are much more frequent than after scarlatinal nephritis. In young children the mortality is high, amounting to at least one third of the cases. Serious symptoms are low arterial tension, the occurrence of uræmia, and effusion into the serous sacs. The persistence of the dropsy after the first month, intense pallor, and a large amount of albumen indicate the possibility of the disease becoming chronic. For some months after the disappearance of the dropsy there may be traces of albumen and a few tube-casts.

In a week or ten days, in a case of scarlatinal nephritis, if the progress is favorable, the dropsy diminishes, the urine increases, the albumen lessens, and by the end of a month the dropsy has disappeared and the urine is nearly free. In very young children the course may be rapid, and I have known the urine to be free from albumen in the fourth week. Other cases are more insidious, and though the dropsy may disappear, the albumen persists in the urine, the anæmia is marked, and the condition becomes chronic or, after several recurrences of the dropsy, improves and complete recovery takes place.

Treatment.—The patient should be in bed and there remain until all traces of the disease have disappeared. As sweating plays such an important part in the treatment, it is well, if possible, to accustom the patient to blankets. He should also be clad in thin Canton flannel.

The diet should consist of milk or butter-milk, gruels made of arrow-root or oat-meal, barley water, and, if necessary, beef tea and chicken broth. It is better, if possible, to confine the patient to a strictly milk diet. As convalescence is established, bread and butter, lettuce, water-cress, grapes, oranges, and other fruits may be given. The return to a meat diet should be gradual.

The patient should drink freely of alkaline mineral waters, ordinary water, or lemonade. The fluids keep the kidneys flushed and wash out the *débris* from the tubes. A useful drink is a drachm of cream of tartar in a pint of boiling water, to which may be added the juice of half a lemon and a little sugar. Taken when cold, this is a pleasant and satisfactory diluent drink.

No remedies, so far as known, control directly the changes which are going on in the kidneys. The indications are: (1) To give the excretory

function of the kidney rest by utilizing the skin and the bowels, in the hope that the natural processes may be sufficient to effect a cure; (2) to meet the symptoms as they arise.

In a case of scarlet fever it may occasionally be possible to avert an attack, the premonitory symptoms of which are marked increase in the arterial tension and the presence of blood coloring matter in the urine (Mahomed). An active saline cathartic may completely relieve this condition.

At the onset, when there is pain in the back or hæmaturia, the dry or wet cups give relief. The latter should not be used in children. Warm poultices are often grateful. In cases which set in with suppression of urine, these measures should be adopted, and in addition the hot bath with subsequent pack, copious diluents, and a free purge. The dropsy is best treated by hydrotherapy—either the hot bath, the wet pack, or the hot-air bath. In children the wet pack is usually satisfactory. It is applied by wringing a blanket out of hot water, wrapping the child in it, covering this with a dry blanket, and then with a rubber cloth. In this the child may remain for an hour. It may be repeated daily. In the case of adults, the hot-air bath or the vapor bath may be conveniently given by allowing the vapor or air to pass from a funnel beneath the bed-clothes, which are raised on a low cradle. More efficient, as a rule, is a hot bath of from fifteen or twenty minutes, after which the patient is wrapped in blankets. The sweating produced by these measures is usually profuse, rarely exhausting, and in a majority of cases the dropsy can in this way be relieved. There are some cases, however, in which the skin does not respond to the baths, and if the symptoms are serious, particularly if uræmia supervenes, jaborandi or its active principle, pilocarpine, may be used. The latter may be given hypodermically, in doses of from a sixth to an eighth of a grain in adults, and from a twentieth to a twelfth of a grain in children from two to ten years. It is a drug to be used with care. I abandoned its employment for many years, after having several cases of serious collapse. Latterly I have resumed its use, often with benefit.

The bowels should be kept open by a morning saline purge; in children the fluid magnesia is readily taken; in adults the sulphate of magnesia may be given by Hay's method, in concentrated form, in the morning, before anything is taken into the stomach. In Bright's disease it not infrequently causes vomiting. The compound powder of jalap, in half-drachm doses, or, if necessary, elaterium may be used. If the dropsy is not extreme, the urine not very concentrated, and uræmic symptoms are not present, the bowels should be kept loose without active purgation. If these measures fail to reduce the dropsy and it has become extreme, the skin may be punctured with a lancet or drained by a small silver canula (Southey's tube), which is inserted beneath it. A fine aspirator needle may be used, and the fluid allowed to drain through a piece of long, narrow rubber tubing into a vessel beneath the bed. If the dyspnœa is marked,

owing to pressure of fluid in the pleuræ, aspiration should be performed. In rare instances the ascites is extreme and may require paracentesis, or a Southey's tube may be inserted and the fluid gradually withdrawn. If uræmic convulsions occur, the intensity of the paroxysms may be limited by the use of chloroform; to an adult a pilocarpine injection should be at once given, and from a robust, strong man twenty ounces of blood may be withdrawn. In children the loins may be dry cupped, the wet pack used, and a brisk purgative given. Bromide of potassium and chloral sometimes prove useful.

Vomiting may be relieved by ice and by restricting the amount of food. Drop doses of creosote, iodine, and carbolic acid may be given. The dilute hydrocyanic acid with bismuth is often effectual.

The question of the use of diuretics in acute Bright's disease is not yet settled. The best diuretic, after all, is water, which may be taken freely with the citrate of potash or the benzoate of soda, salts which are held to favor the conversion of the urates into less irritating and more easily excreted compounds. Digitalis and strophanthus are useful diuretics, and may be employed without risk when the arterial tension is low and the cardiac impulse is not forcible. I have never seen any injurious effects from their employment after the early symptoms had lessened in intensity.

For the persistent albuminuria, I agree with Roberts and Rosenstein that we have no remedy of the slightest value. Nothing indicates more clearly our helplessness in controlling kidney metabolism than inability to meet this common symptom. Astringents, alkalies, nitroglycerin, and mercury have been recommended.

For the anæmia always associated with acute Bright's disease iron should be employed. It should not be given until the acute symptoms have subsided. In the adult it may be used in the form of the perchloride in increasing doses, as convalescence proceeds. In children, the syrup of the iodide of iron or the syrup of the phosphate of iron are better preparations. The dilatation of the heart is best treated with digitalis, strophanthus, and strychnia.

In the convalescence from acute Bright's disease, care should be taken to guard the patient against cold. The diet should still consist chiefly of milk and a return to mixed food should be gradual. A change of air is often beneficial, particularly a residence in a warm, equable climate.

VI. CHRONIC BRIGHT'S DISEASE.

Here, too, in all forms we deal with a diffuse process, involving epithelial, interstitial, and glomerular tissues. Clinically two groups are recognized—(*a*) the chronic parenchymatous nephritis, which follows the acute attack or comes on insidiously, is characterized by marked dropsy, and post mortem by the *large white kidney*. In the later stages of this

process the kidney may be smaller—a condition known as the *small white kidney*; (*b*) chronic interstitial nephritis, in which dropsy is not common and the cardio-vascular changes are pronounced. Delafield recognizes a chronic diffuse nephritis with exudation and a chronic productive diffuse nephritis without exudation, the latter corresponding to the contracted kidney of authors.

The amyloid kidney is usually spoken of as a variety of Bright's disease, but in reality it is a degeneration which may accompany any form of nephritis.

Chronic Parenchymatous Nephritis

(*Chronic Desquamative and Chronic Tubal Nephritis; Chronic Diffuse Nephritis with Exudation*).

Etiology.—In many cases the disease follows the acute nephritis of cold, scarlet fever, or pregnancy. More frequently than is usually stated the disease has an insidious onset and occurs independently of any acute attack. The fevers may play an important *rôle* in certain of these cases. Rosenstein, Bartels, and, in this country, I. E. Atkinson have laid special stress upon malaria as a cause. No instance of the kind has fallen under my observation during the past seven years, in which time several hundred cases of malaria have been under my treatment. Beer and alcohol are believed to lead to this form of nephritis. In chronic suppuration, syphilis, and tuberculosis the diffuse parenchymatous nephritis is not uncommon, and is usually associated with amyloid disease. Males are rather more subject to the affection than females. It is met with most commonly in young adults, and is by no means infrequent in children as a sequence of scarlatinal nephritis.

Morbid Anatomy.—Several varieties of this form have been recognized. The most common is the *large white kidney* of Wilks, in which the organ is enlarged, the capsule is thin, and the surface white with the stellate veins injected. On section the cortex is swollen and yellowish white in color, and often presents opaque areas. The pyramids may be deeply congested. On microscopical examination it is seen that the epithelium is granular and fatty, and the tubules of the cortex are distended, and contain tube-casts. Hyaline changes are also present in the epithelial cells. The glomeruli are large, the capsules thickened, the capillaries show hyaline changes, and the epithelium of the tuft and of the capsule is extensively altered. The interstitial tissue is everywhere increased, though not to an extreme degree.

The second variety of this form results from the gradual increase in the connective tissue and the subsequent shrinkage, forming what is called the *small white kidney* or the pale granular kidney. It is doubtful whether this is always preceded by the large white kidney. Some observers hold that it may be a primary independent form. The capsule is thick-

ened and the surface is rough and granular. On section the resistance is greatly increased, the cortex is reduced and presents numerous opaque white or whitish-yellow foci, consisting of accumulations of fatty epithelium in the convoluted tubules. This combination of contracted kidney with the areas of marked fatty degeneration has given the name of small granular, fatty kidney to this form. The interstitial changes are marked, many of the glomeruli are destroyed, the degeneration of epithelium in the convoluted tubules is wide-spread, and the arteries are greatly thickened.

Belonging to this chronic tubal nephritis is a variety known as the *chronic hæmorrhagic nephritis*, in which the organs are enlarged, yellowish white in color, and in the cortex are many brownish-red areas, due to hæmorrhage into and about the tubes. In other respects the changes are identical with those in the large white kidney.

Of changes in the other organs the most marked are thickening of the blood-vessels and hypertrophy of the left heart.

Symptoms.—Following an acute nephritis, the disease may present, in a modified way, the symptoms of that affection. In many cases it sets in insidiously, and after an attack of dyspepsia or a period of failing health and loss of strength the patient becomes pale and puffiness of the eyelids or swollen feet are noticed in the morning.

The symptoms are as follows: The urine is, as a rule, diminished in quantity, often scanty. It has a dirty-yellow, sometimes smoky, color and is turbid from the presence of urates. On standing, a heavy sediment falls, in which are found numerous tube-casts of various forms and sizes, hyaline, both large and small, epithelial, granular, and fatty casts. Leucocytes are abundant; red blood-corpuscles are frequently met with, and epithelium from the kidneys and pelves. The albumen is abundant and may amount to one half or one third of the urine boiled. It is more abundant in the urine passed during the day. The specific gravity may be high in the early stages—from 1·020 to 1·025—though in the later stages it is lower. The urea is always reduced in quantity.

Dropsy is a marked and obstinate symptom of this form of Bright's disease. The face is pale and puffy, and in the morning the eyelids are œdematous. The anasarca is general, and there may be involvement of the serous sacs. In these chronic cases associated with large white kidney there is often a distinctive appearance in the face; the complexion is pasty, the pallor marked, and the eyelids are œdematous. The dropsy is peculiarly obstinate. Uræmic symptoms are common, though convulsions are perhaps less frequent than in the interstitial nephritis.

The tension of the pulse is usually increased; the vessels ultimately become stiff and the heart hypertrophied, though there are instances of this form of nephritis in which the heart is not enlarged. The aortic second sound is accentuated. Retinal changes though less frequent than in the chronic interstitial nephritis, occur in a considerable number of cases.

Gastro-intestinal symptoms are common. Vomiting is frequently a distressing and serious symptom, and diarrhœa may be profuse. Ulceration of the colon may occur and prove fatal.

It is sometimes impossible to determine, even by the most careful examination of the urine or by analysis of the symptoms, whether the condition of the kidney is that of the large white or of the small white form. In cases, however, which have lasted for several years, with the progressive increase in the renal connective tissue and the cardio-vascular changes, the clinical picture may approach, in certain respects, that of the contracted kidney. The urine is increased, with low specific gravity. It is often turbid, may contain traces of blood, the tube-casts are numerous and of every variety of form and size, and the albumen is abundant. Dropsy is usually present, though not so extensive as in the early stages.

The *prognosis* is extremely grave. In a case which has persisted for more than a year recovery rarely takes place. Death is caused either by great effusion with œdema of the lungs, by uræmia, or by secondary inflammation of the serous membranes. Occasionally in children, even when the disease has persisted for two years, the symptoms disappear and recovery takes place.

Treatment.—Essentially the same treatment should be carried out as in acute Bright's disease. Milk or butter-milk should constitute the chief article of food. The dropsy should be treated by hydrotherapy. Iron preparations should be given freely. The acetate of potash and digitalis are useful in increasing the flow of urine. Basham's mixture given in plenty of water will be found beneficial.

Chronic Interstitial Nephritis.

(*Contracted Kidney; Granular Kidney; Cirrhosis of the Kidney; Gouty Kidney; Renal Sclerosis*).

Sclerosis of the kidney is met with (*a*) as an occasional sequence of the large white kidney, forming the so-called pale granular or secondary contracted kidney; (*b*) as an independent primary affection; (*c*) as a sequence of arterio-sclerosis.

Etiology.—The primary form is chronic from the outset, and is a slow, creeping degeneration of the kidney substance—in many respects only an anticipation of the gradual changes which take place in the organ in extreme old age. In many cases no satisfactory cause can be assigned. In others there are hereditary influences, as in the remarkable family studied by Dickenson, in which a pronounced tendency to chronic Bright's disease occurred in four generations. Families in which the arteries tend to degenerate early are more prone to interstitial nephritis. Syphilis is held by some to be a cause. Alcohol probably plays an important part, particularly in conjunction with other factors. Dietetic influences are at work in many cases. Some believe excessive use of meat is injurious, since it

increases the materials out of which uric acid is formed. By many a functional disorder of the liver, leading to lithæmia, is regarded as the most efficient factor. It is quite possible that in persons who habitually eat and drink too much the work thrown upon this organ is excessive, and the elaboration of certain materials so defective that in their excretion from the general circulation they irritate the kidneys.

Actual gout, which in England is a common cause of interstitial nephritis, is not an important factor here. On the other hand, the nutritional disorder known as lithæmia is very common, either with or without dyspepsia. Lead, as is well known, may produce renal sclerosis, but it is a minor factor in comparison with other causes. It is doubtful if climate has any influence. Purdy regards the cold, moist regions of the Northeastern States as specially favorable to the disease.

Among factors which may account for the prevalence of chronic Bright's disease in the better classes in this country may be mentioned the intense worry and strain of business, combined, as they often are, with habits of hurried and over eating and a lack of proper exercise. Males are more commonly attacked than females. Under twenty-five years of age it is a rare disease; between twenty-five and forty a few well-marked cases occur; between forty and sixty it is common.

Morbid Anatomy.—The kidneys are usually small, and together may weigh no more than an ounce and a half. The capsule is thick and adherent; the surface of the organ irregular and covered with small nodules, which have given to it the name of granular kidney. In stripping off the capsule, portions of the kidney substance are removed. Small cysts are frequently seen on the surface. The color is usually reddish, often a very dark red. On section the substance is tough and resists cutting; the cortex is thin and may measure no more than a couple of millimetres. The pyramids are less wasted. The small arteries are greatly thickened and stand out prominently. The fat about the pelvis is greatly increased.

Microscopically there is seen a marked increase in the connective tissue and degeneration and atrophy of the secreting structures, glomerular and tubal, the former being most predominant and giving the main characters to the lesion. The following are the most important changes:

(*a*) An increase in the fibrous elements, widely distributed throughout the organ, but more advanced in the cortex, particularly in the tissue between the medullary rays. In the pyramids the distribution of new growth is less patchy and more diffuse. In the early stages of the process there is a small-celled infiltration between the tubes and around the glomeruli, and finally this becomes fibrillated and is seen encircling the tubules and Bowman's capsules, around the latter often forming concentric layers.

(*b*) The changes in the glomeruli are striking, and in advanced cases a very considerable number of them have undergone complete atrophy and are represented as densely encapsulated hyaline structures. The atrophy is partly due to changes in the capillary walls and multiplication of cells

between the loops, partly to extensive hyaline degeneration, and in part, no doubt, to the alterations in the afferent vessels. The normal glomeruli usually show some thickening of the capsule and increase in the cells of the tufts.

(*c*) The tubules show changes in the epithelium, which vary a good deal in different localities. Where the connective-tissue growth is most advanced they are greatly atrophied and the epithelium may be represented by small cubical cells. In other instances the epithelium has entirely disappeared. On the other hand, in the regions represented by the projecting granules the tubules are usually dilated, and the epithelium shows hyaline, fatty, and granular changes. Very many of them contain dark masses of epithelial *débris* and tube-casts. In the interstitial tissue and in the tubules there may be pigmentary changes due to hæmorrhage. The dilatation of the tubules may reach an extreme grade, forming definite cysts.

(*d*) The arteries show an advanced sclerosis. The intima is greatly thickened and there are changes in the adventitia and in the media, consisting in increase in the thickness due to proliferation of the connective tissue, in the latter coat at the expense of the muscular elements.

The view most generally entertained at present is that the essential lesion is in the secreting tissues of the tubules and the glomeruli, and that the connective-tissue overgrowth is secondary to this. Greenfield holds that the primary change is in most instances in the glomeruli, to which both the degeneration in the epithelium of the convoluted tubules and the increase in the intertubular connective tissue are secondary.

Associated with contracted kidney are general arterio-sclerosis and hypertrophy of the heart. The changes in the arteries have already been described in the section on arterio-sclerosis. The hypertrophy of the heart is almost constant. I do not remember ever to have seen a well-marked instance of contracted kidney without some hypertrophy of the left ventricle, and the enlargement may reach an extreme grade. The variations depend, no doubt, in part upon the extent of the diffuse arterial degeneration, and there are instances in which the term *cor bovinum* may be applied to the enlarged organ. In such cases the hypertrophy is not confined to the left ventricle, but involves the entire heart. The explanation of this hypertrophy has been much discussed. It was at first held to be due to the increased work thrown upon the organ in driving the impure blood through the capillary system. Basing his opinion upon the supposed muscular increase in the smaller arteries, Johnson regarded the hypertrophy as effort to overcome a sort of stop-cock action of these vessels, which, under the influence of the irritating ingredient in the blood, contracted and increased greatly the peripheral resistance. Traube believed that the obliteration of a large number of capillary territories in the kidney materially raised the arterial pressure, and in this way led to the hypertrophy of the heart; an additional factor, he thought, was the

diminished excretion of water, which also heightened the pressure within the blood-vessels.

In our present knowledge the most satisfactory explanation is that given by Cohnheim, which is thus clearly and succinctly put by Fagge: "He gives reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilatation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings . . . upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. But in order that such a quantity of blood should pass through the restricted capillary area now open to it, an excessive pressure must obviously be necessary. This can be brought to bear only by the exertion of more than the normal degree of force on the part of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure and for the cardio-vascular changes that are secondary to it."

Symptoms.—Perhaps a majority of the cases are latent, and are not recognized until the occurrence of one of the serious or fatal complications. Even an advanced grade of contracted kidney may be compatible with great mental and bodily activity. There may have been no symptoms whatever to suggest to the patient the existence of a serious malady. In other cases the general health is disturbed. The patient complains of lassitude, is sleepless, has to get up at night to micturate; the digestion is disordered, the tongue is furred; there are complaints of headache, failing vision, and breathlessness on exertion.

So complex and varied is the clinical picture of chronic Bright's disease that it will be best to consider the symptoms under the various systems.

Urinary System.—The amount of urine is usually increased, and from two to four litres may be passed. Frequently the patient has to get up two or three times during the night to empty the bladder, and there is increased thirst. It is for these symptoms occasionally that relief is sought. It is to be remembered, however, that frequent micturition at night may be associated with irritability of the prostate and, in certain cases, with hyperacidity of the urine. The secretion is clear, the mucus cloud is well marked, but there is no definite sediment. The color is a light yellow, and the specific gravity ranges from 1·005 to 1·012. Traces of albumen are found, but may be absent at times, particularly in the early morning urine. It is often simply a slight cloudiness, and may be apparent only

slight leakage may represented by the constant presence of a few red cells, may be present early in the disease and persist for years. In other instances there may be, especially after exercise, flecks of blood in an otherwise pale smoky urine

with the more delicate tests. The sediment is scanty, and in it a few hyaline or granular casts are found. The quantity of the solid constituents of the urine is, as a rule, diminished, though in some instances the urea may be excreted in full amount. In attacks of dyspepsia or bronchitis, or in the later stages when the heart fails, the quantity of albumen may be greatly increased and the urine diminished. Occasionally blood occurs in the urine, and there may even be hæmaturia (S. West).

Circulatory System.—The pulse is hard, the tension increased, and the vessel wall, as a rule, thickened. As already mentioned, a distinction must be made between increased tension and thickening of the arterial wall. The tension may be plus in a normal vessel, but in chronic Bright's disease it is more common to find the tension is increased and the artery stiff.

A pulse of increased tension has the following characters: (*a*) It is hard and incompressible, requiring a good deal of force to overcome it; (*b*) it may be impossible to obliterate the pulse wave by any pressure on the vessel; (*c*) it is persistent, and in the intervals between the beats the vessel feels full and can be rolled beneath the finger. These characters may be present in a vessel the walls of which are little, if at all, increased in thickness. To estimate the latter the pulse wave should be obliterated in the radial, and the vessel wall felt beyond it. In a perfectly normal vessel the arterial coats, under these circumstances, cannot be differentiated from the surrounding tissue; whereas, if thickened, the vessel can be rolled beneath the finger. Persistent high tension is one of the earliest and most important symptoms of interstitial nephritis. The cardiac features are equally important, though often less obvious. Hypertrophy of the left ventricle occurs to overcome the resistance offered in the arteries. The enlargement of the heart ultimately becomes more general. The apex is displaced downward and to the left; the impulse is forcible and may be heaving. In elderly persons with emphysema, the displacement of the apex may not be evident. The first sound at the apex may be duplicated; more commonly the second sound at the aortic cartilage is accentuated, a very characteristic sign of increased tension. The sound in extreme cases may have a bell-like quality. In many cases a systolic murmur develops at the apex, probably as a result of relative insufficiency. It may be loud and transmitted to the axilla. Finally the hypertrophy fails, the heart becomes dilated, gallop rhythm is present, and the general condition is that of a chronic heart-lesion.

Respiratory System.—Sudden œdema of the glottis may occur. Effusion into the pleuræ or sudden œdema of the lungs may prove fatal. Acute pleurisy and pneumonia are not uncommon. Bronchitis is a frequent accompaniment, particularly in the winter. Sudden attacks of oppressed breathing, particularly at night, are not infrequent. This is often a uræmic symptom, but is sometimes cardiac. The patient may sit up in bed and gasp for breath, as in true asthma. Cheyne-Stokes breath-

ing may be present, most commonly toward the close, but the patient may be walking about and even attending to his occupation.

Digestive System.—Dyspepsia and loss of appetite are common. Severe and uncontrollable vomiting may be the first symptom. This is usually regarded as a manifestation of uræmia, but it may be present without any other indications, and I have known it to prove fatal without any suspicion that chronic Bright's disease was present. Severe and even fatal diarrhœa may develop. The tongue may be coated and the breath heavy and urinous.

Nervous System.—Various cerebral manifestations have already been mentioned under uræmia, and they are among the most important of the features of chronic Bright's disease. Cerebral apoplexy is closely related to interstitial nephritis. The hæmorrhage may take place into the meninges or the cerebrum. It is usually associated with marked changes in the vessels. Neuralgias, in various regions, are not uncommon.

Special Senses.—Troubles in vision may be the first symptom of the disease. It is remarkable in how many cases of interstitial nephritis the condition is diagnosed first by the ophthalmic surgeon. The flame-shaped hæmorrhages are the most common. Less frequent is diffuse retinitis or papillitis. Sudden blindness may supervene without retinal changes—uræmic amaurosis. Auditory troubles are by no means infrequent in chronic Bright's disease. Ringing in the ears, with dizziness, is not uncommon. Various forms of deafness may occur.

Skin.—Œdema is not common in interstitial nephritis. Slight puffiness of the ankles may be present, but in a majority of the cases dropsy does not supervene. When extensive, it is almost always the result of gradual failure of the hypertrophied heart. The skin is often dry and pale, and sweats are not common. In some instances the sweat may deposit a white frost of urea on the surface of the skin. Eczema is a common accompaniment of chronic interstitial nephritis. Tingling of the fingers or numbness and pallor—the dead fingers—are not, as some suppose, in any way peculiar to Bright's disease. Intolerable itching of the skin may be present, and cramps in the muscles are by no means rare.

Hæmorrhages are not infrequent; thus, epistaxis may occur and prove serious. Purpura may develop. Broncho-pulmonary hæmorrhages are said, by some French writers, to be common, but no instance of it has come under my observation. Ascites is rare except in association with cirrhosis of the liver.

Diagnosis.—The autopsy often discloses the true nature of the disease, one of the many intercurrent affections of which may have proved fatal. The early stages of interstitial nephritis are not recognizable. In a patient with increased pulse tension (particularly if the vessel wall is sclerotic), with the apex beat of the heart dislocated to the left, the second aortic sound ringing and accentuated, the urine abundant and of low specific gravity, with a trace of albumen and an occasional hyaline or

granular cast, the diagnosis of interstitial nephritis may be safely made. Of all the indications, that offered by the pulse is the most important. Persistent high tension with thickening of the arterial wall in a man under fifty means that serious mischief has already taken place, that cardio-vascular changes are certainly, and renal most probably, present. It is important in the diagnosis of this condition not to rest content with a single examination of the urine. Both the evening and the morning secretion should be studied. The sediment should be collected in a conical glass, and in looking for tube-casts a large surface should be examined with a tolerably low power and little light. The arterio-sclerotic kidney may exist for a long time without the occurrence of albumen, or the albumen may be in very small quantities. In many cases it is impossible to differentiate the primary interstitial nephritis from an arterio-sclerotic kidney, nor clinically is it of any special value so to do. In persons under forty, with very high tension, great thickening of the superficial arteries, and marked hypertrophy of the heart, the renal are more likely to be secondary to the arterial changes.

Prognosis.—Chronic Bright's disease is an incurable affection, and the anatomical conditions on which it depends are quite as much beyond the reach of medicines as wrinkled skin or gray hair. Interstitial nephritis, however, is compatible with the enjoyment of life for many years, and it is now universally recognized that increased tension, thickening of the arterial walls, and polyuria with a small quantity of albumen, neither doom a man to death within a short time nor necessarily interfere with the pursuits of an active life so long as proper care be taken. I know patients who have had high tension and a little albumen in the urine with hyaline casts for ten, twelve, and, in one instance, fifteen years. Serious indications are the development of uræmic symptoms, dilatation of the heart, the onset of serous effusions, the development of Cheyne-Stokes breathing, persistent vomiting, and diarrhœa.

Treatment.—Patients without local indications or in whom the condition has been accidentally discovered should so regulate their lives as to throw the least possible strain upon heart, arteries, and kidneys. A quiet life without mental worry, with gentle but not excessive exercise, and residence in an equable climate, should be recommended. In addition they should be told to keep the bowels regular, the skin active by a daily tepid bath with friction, and the urinary secretion free by drinking daily a definite amount of either distilled water or some pleasant mineral water. Alcohol should be strictly prohibited. Tea and coffee are allowable.

The diet should be light and nourishing, and the patient should be warned not to eat excessively, and not to take meat more than once a day. Care in food and drink is probably the most important element in the treatment of these early cases.

A patient in good circumstances may be urged to go away during the winter months, or, if necessary, to move altogether to a warm equable cli-

mate, like that of southern California. There is no doubt of the value in these cases of removal from the changeable, irregular weather which prevails in the temperate regions from November until April.

At this period medicines are not required unless for certain special symptoms. Patients derive much benefit from an annual visit to certain mineral springs, such as Poland, Bedford, Saratoga, in this country, and Vichy and others in Europe. Mineral waters have no curative influence upon chronic Bright's disease; they simply help the interstitial circulation and keep the drains flushed. In this early stage, when the patient's condition is good, the tension not high, and the quantity of albumen small, medicines are not indicated, since no remedies are known to have the slightest influence upon the progress of the disease. Sooner or later symptoms arise which demand treatment. Of these the following are the most important:

(*a*) *Greatly Increased Arterial Tension.*—It is to be remembered that a certain increase of tension is not only necessary but unavoidable in chronic Bright's disease, and probably the most serious danger is too great lowering of the blood tension. The happy medium must be sought between such heightened tension as throws a serious strain upon the heart and risks rupture of the vessels and the low tension which, under these circumstances, is specially liable to be associated with serous effusions. In cases with persistent high tension the diet should be light, an occasional saline purge should be given, and sweating promoted by means of hot air or the hot bath. If these measures do not suffice, nitroglycerin may be tried, beginning with one minim of the one per cent. solution three times a day, and gradually increasing the dose if necessary. Patients vary so much in susceptibility to this drug that in each case it must be tested, the limit of dosage being that at which the patient experiences the physiological effect. As much as ten minims of the one per cent. solution may be given three times a day. In many cases I have given it in much larger doses for weeks at a time. I have never seen any ill effects from it. If the dose is excessive the patients complain at once of flushing or headache. Its use may be kept up for six or seven weeks, then stopped for a week and resumed. Its value is seen not only in the reduction of the tension, but also in the striking manner in which it relieves the headache, dizziness, and dyspnœa.

(*b*) More or less *anæmia* is present in advanced cases, which is best met by the use of iron. Weir Mitchell, who has had a unique experience in certain forms of chronic Bright's disease, gives the tincture of the perchloride of iron in large doses—from half a drachm to a drachm three times a day. He thinks that it not only benefits the anæmia, but that it also is an important means of reducing the arterial tension.

(*c*) Many patients with Bright's disease present themselves for treatment with signs of cardiac dilatation; there is a gallop rhythm or the heart sounds have a fœtal character, the breath is short, the urine scanty and

highly albuminous, and there are signs of local dropsy. In these cases the treatment must be directed to the heart. A morning dose of salts or calomel may be given, and digitalis in ten-minim doses, three or four times a day. Strychnia may be used with benefit in this condition. In some instances other cardiac tonics may be necessary, but as a rule the digitalis acts promptly and well.

(*d*) *Uræmic Symptoms.*—Even before marked manifestations are present there may be extreme restlessness, mental wandering, a heavy, foul breath, and a coated tongue. Headache is not often complained of, though intense frontal headache may be an early symptom of uræmia. In this condition, too, the patient may complain of palpitation, feelings of numbness, and sometimes nocturnal cramps. For these symptoms the saline purgatives should be ordered, and hot baths, so as to induce copious sweating. Nitroglycerin also may be freely used to reduce the tension. For the uræmic convulsions, if severe, inhalations of chloroform may be used. If the patient is robust and full-blooded, from twelve to twenty ounces of blood should be removed. The patient should be freely sweated, and if the convulsions tend to recur chloral may be given, either by the mouth or per rectum, or, better still, morphia. Uræmic coma must be treated by active purgation, and sweating should be promoted by the use of pilocarpine or the hot bath. For the restlessness and delirium morphia is indispensable. Since its recommendation in uræmic states some years ago, by Stephen MacKenzie, I have used this remedy extensively and can speak of its great value in these cases. I have never seen ill effects or any tendency to coma follow.

VII. AMYLOID DISEASE.

Amyloid (lardaceous or waxy) degeneration of the kidneys is simply an event in the process of chronic Bright's disease, most commonly in the chronic parenchymatous nephritis following fevers or of cachectic states. It has no claim to be regarded as one of the varieties of Bright's disease. The affection of the kidneys is generally a part of a wide-spread amyloid degeneration occurring in prolonged suppuration, as in disease of the bone, in syphilis, tuberculosis, and less commonly in association with leukæmia, lead poisoning, and gout.

Anatomically the amyloid kidney is large and pale, the surface smooth, and the venæ stellatæ well marked. On section the cortex is large and may show a peculiar glistening, infiltrated appearance, and the glomeruli are very distinct. The pyramids, in striking contrast to the cortex, are of a deep red color. A section soaked in dilute tincture of iodine shows spots of a walnut or mahogany brown color. The Malpighian tufts and the straight vessels may be most affected. In lardaceous disease of the kidneys the organs are not always enlarged. They may be normal in size or

small, pale, and granular. The amyloid change is first seen in the Malpighian tufts, and then involves the afferent and efferent vessels and the straight vessels. It may be confined entirely to them. In later stages of the disease the tubules are affected, chiefly the membrane, rarely, if ever, the cells themselves. In addition, the kidneys always show signs of diffuse nephritis. The Bowman's capsules are thickened, there may be glomerulitis, and the tubal epithelium is swollen, granular, and fatty.

Symptoms.—The renal features alone may not indicate the presence of this degeneration. Usually the associated condition gives a hint of the nature of the process. The urine, as a rule, shows important changes; the quantity is increased, and it is pale, clear, and of low specific gravity. The albumen is usually abundant, but it may be scanty, and in rare instances absent. Possibly the variations in the situation of the amyloid changes may account for this, since albumen is less likely to be present when the change is confined to the vasæ rectæ. In addition to ordinary albumen, globulin may be present. The tube-casts are variable, usually hyaline, often fatty or finely granular. Occasionally the amyloid reaction can be detected in the hyaline casts. Dropsy is present in many instances, particularly when there is much anæmia or profound cachexia. It is not, however, an invariable symptom, and there are cases in which it does not develop.

Increased arterial tension and cardiac hypertrophy are not usually present, except in those cases in which amyloid degeneration occurs in the secondary contracted kidney; under which circumstances there may be uræmia and retinal changes, which, as a rule, are not met with in other forms.

Diagnosis.—By the condition of the urine alone it is not possible to recognize amyloid changes in the kidney. Usually, however, there is no difficulty, since the Bright's disease comes on in association with syphilis, prolonged suppuration, disease of the bone, or tuberculosis, and there is evidence of enlargement of the liver and spleen. A suspicious circumstance is the existence of polyuria with a large amount of albumen in the urine, or when, in these constitutional affections, a large quantity of clear, pale urine is passed, even without the presence of albumen.

The prognosis depends rather on the condition with which the nephritis is associated. As a rule it is grave.

The treatment of the condition is that of chronic Bright's disease.

VIII. PYELITIS

(Consecutive Nephritis; Pyelonephritis; Pyonephrosis).

Definition.—Inflammation of the pelvis of the kidney and the conditions which result from it.

Etiology.—Pyelitis is induced by many causes, among which the following are the most important: (*a*) The irritation of calculi—a very

frequent cause. (*b*) Tubercle. (*c*) The infectious pyelitis which develops in typhoid fever, pneumonia, scarlet fever, diphtheria, small-pox, and other fevers. Here an acute inflammation of the pelvis of the kidney may occur, sometimes hæmorrhagic in character, more frequently diphtheritic. (*d*) The presence of decomposing urine, following pressure upon the ureter by tumors or bladder-disease. By far the most frequent form of pyelitis is that which is consecutive to cystitis, from whatever cause. In these cases the inflammation may not be confined to the pelvis, but pass to the kidney, inducing pyelonephritis. (*e*) Occasional causes are cancer, hydatids, the ova of certain parasites, and, according to some, the irritation of the saccharine urine of diabetes, and the irritation of turpentine or cubebs. (*f*) A primary pyelitis or pyelonephritis has been described as coming on after cold or overexertion, but such cases are extremely rare.

Morbid Anatomy.—In the early stages of pyelitis the mucous membrane is turbid, somewhat swollen, and may show ecchymoses. The urine in the pelvis is cloudy, and, on examination, numbers of epithelial cells are seen. In the form associated with the infectious fevers there is usually a grayish pseudo-membrane, either limited to an infundibulum or involving a great part of the pelvis.

In the calculous pyelitis there may be only slight turbidity of the membrane, which has been called by some catarrhal pyelitis. More commonly the mucosa is roughened, grayish in color, thick, and, on microscopical examination, the tissues are seen to be infiltrated with leucocytes. Under these circumstances there is almost always more or less dilatation of the calyces and flattening of the papillæ. Following this condition there may be (*a*) extension of the suppurative process to the kidney itself, forming a pyelonephritis; (*b*) a gradual dilatation of the calyces with atrophy of the kidney substance, and finally the production of the condition of pyonephrosis, in which the entire organ is represented by a sac of pus with or without a thin shell of renal tissue. (*c*) After the kidney structure has been destroyed by suppuration, and the obstruction at the orifice of the pelvis persists, the fluid portions may be absorbed, the pus becomes inspissated, so that the organ is represented by a series of sacculi containing grayish, putty-like masses, which may become impregnated with lime salts.

Tuberculous pyelitis, as already described, usually starts upon the apices of the pyramids, and may at first be limited in extent. Ultimately the condition produced may be similar to that of calculous pyelitis. Pyonephrosis is quite as frequent a sequence, while the final transformation of the pus into a putty-like material impregnated with salts, forming the so-called scrofulous kidney, is even commoner.

The pyelitis consecutive to cystitis is usually bilateral, and the kidney is apt to be involved, forming the so-called *surgical kidney*—acute suppurative nephritis. There are lines of suppuration extending along the pyramids, or small abscesses in the cortex, often just beneath the capsule;

or there may be wedge-shaped abscesses. The pus organisms either pass up the tubules or, as Steven has shown, pass by the lymphatics.

Symptoms.—The forms associated with the fevers rarely cause any symptoms, even when the process is extensive. In mild grades there is pain in the back or there may be tenderness on deep pressure on the affected side. The urine is turbid, contains a few mucous and pus cells, and occasionally blood-corpuscles. The urine is acid, and there may be a trace of albumen.

Before the condition of pyuria is established there may be attacks of pain on the affected side (not amounting to the severe agony of renal colic), rigors, high fever, and sweats. Under these circumstances the urine, which may have been clear, becomes turbid or smoky from the presence of blood, and may contain large numbers of mucus cells and transitional epithelium. These cases are not common, but I have twice had opportunity of studying such attacks for a prolonged period. In one patient the occurrence of the rigor and fever could sometimes be predicted from the change in the condition of the urine. Such cases occur, I believe, in association with calculi in the pelvis.

The statement is not infrequently made that the epithelium in the urine in pyelitis is distinctive and characteristic. This is erroneous, as may be readily demonstrated by comparing scrapings of the mucosa of the pelvis and of the bladder. In both the epithelium belongs to what is called the transitional variety, and in both regions the same conical, fusiform and irregular cells with long tails are found.

When the pyelitis, whether calculous or tuberculous, has become chronic and suppurative, the symptoms are:

(1) *Pyuria.*—The pus is in variable amount, and may be intermittent. Thus, as is often the case when only one kidney is involved, the ureter may be temporarily blocked, normal urine is passed for a time, and then there is a sudden outflow of the pent-up pus and the urine becomes purulent. Coincident with this retention, a tumor mass may be felt on the side affected. The pus has the ordinary characters, but the transitional epithelium is not so abundant at this stage and comes from the bladder or from the pelvis of the healthy side. Occasionally in rapidly advancing pyelonephritis portions of the kidney tissue, particularly of the apices of the pyramids, may slough away and appear in the urine; or, as in a remarkable specimen shown to me by Tyson, solid cheesy moulds of the calyces are passed. Casts from the kidney tubules are sometimes present. The reaction of the urine is at first acid, and may remain so even when the pus is passed in large quantities. If it remains any time in the bladder or if cystitis exists it becomes ammoniacal. Micturition may be very frequent and irritability of the bladder may be present.

(2) Intermittent fever associated with rigors is usually present in cases of suppurative pyelitis. The chills may recur at regular intervals, and the cases are often mistaken for malaria. Owen-Rees called attention to

the frequent occurrence of these rigors, which form a characteristic feature of both calculous and tuberculous pyelitis. Ultimately the fever assumes a hectic type and the rigors may cease.

(3) The general condition of the patient usually indicates prolonged suppuration. There is more or less wasting with anæmia and a progressive failure of health. Secondary abscesses may develop and the clinical picture becomes that of pyæmia. In some instances, particularly of tuberculous pyelitis, the clinical course may resemble that of typhoid fever. There are instances of pyuria recurring, at intervals, for many years without impairment of the bodily vigor.

(4) Physical examination in chronic pyelitis usually reveals tenderness on the affected side or a definite swelling, which may vary much in size and ultimately attain large dimensions if the kidney becomes enormously distended, as in pyonephrosis.

(5) Occasionally nervous symptoms, which may be associated with dyspnœa, supervene, or the termination may be by coma, not unlike that of diabetes. These have been attributed to the absorption of the decomposing materials in the urine, and has been called ammoniæmia. A form of paraplegia has been described in connection with some cases of abscess of the kidney, but whether due to a myelitis or to a peripheral neuritis has not yet been determined.

In suppurative nephritis or surgical kidney following cystitis, the patient complains of pain in the back, the fever becomes high, irregular, and associated with chills, and in acute cases a typhoid state develops in which death occurs.

Diagnosis.—Between the tuberculous and the calculous forms of pyelitis it may be difficult or impossible to distinguish, except by the detection of tubercle bacilli in the pus. This has been done on several occasions, but many slides must be examined, for the bacilli are usually scanty. From perinephric abscess pyonephrosis is distinguished by the more definite character of the tumor, the absence of œdematous swelling in the lumbar region, and, most important of all, the history of the case. The urine, too, in perinephric abscess may be free from pus. There are cases, however, in which it is difficult indeed to make a satisfactory diagnosis. A patient whom I saw with Fussell had had cystitis through her pregnancy, subsequently pus in the urine for several months, and then a large fluctuating abscess developed in the right lumbar region. It did not seem possible, either before or during the operation, to determine whether the case was a simple pyonephrosis or whether there had been a perinephric abscess caused by the pyelitis.

Suppurative pyelitis and cystitis are frequently confounded. I have known three instances of the former in which perineal section was performed on the supposition of the existence of an intractable cystitis. The two conditions may, of course, coexist and prove puzzling, but the history, the acid character of the pus in many instances, the less frequent

occurrence of ammoniacal decomposition, the local signs in one lumbar region, and the absence of pain in the bladder should be sufficient to differentiate the affections. In women, by catheterization of the ureters, it may be definitely determined whether the pus comes from the kidneys or from the bladder.

Prognosis.—Cases coming on during the fevers usually recover. Tuberculous pyelitis may terminate favorably by inspissation of the pus and conversion into a putty-like substance with deposition of lime salts. When pyonephrosis develops the dangers are increased. Perforation may occur, the patient may be worn out by the hectic fever, or amyloid disease may develop.

Treatment.—In mild cases fluids should be taken freely, particularly the alkaline mineral waters, to which the citrate of potash may be added.

The treatment of the calculous form will be considered later. Practically there are no remedies which have much influence upon the pyuria. Astringents in no way control the discharge, nor have I seen the slightest benefit from buchu, copaiba, sandal-wood oil, or uva ursi. Tonics should be given, a nourishing diet, and milk and butter-milk may be taken freely. When the tumor has formed or even before it is perceptible, if the symptoms are serious and severe, the kidney should be explored, and, if necessary, nephrotomy should be performed.

IX. HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calyces of the kidney with atrophy of its substance, caused by the accumulation of non-purulent fluids the result of obstruction.

Etiology.—The condition may be congenital, owing to some abnormality in the ureter or urethra. The tumor produced may be large enough to retard labor. Sometimes it is associated with other malformations. There is a condition of moderate dilatation, apparently congenital, which is not connected with any obstruction in the ducts. A case of the kind was shown at the Philadelphia Pathological Society by Daland.

In some instances there has been contraction or twisting of the ureter, or it has been inserted into the kidney at an acute angle or at a high level. In adult life the condition may be due to lodgement of a calculus, or to a cicatricial stricture following ulcer.

New growths, such as tubercle or cancer, occasionally induce hydronephrosis. More commonly, pressure upon the ureter from without, particularly tumors of the ovaries and uterus. Occasionally cicatricial bands compress the ureter. Obstruction within the bladder may result from cancer, from hypertrophy of the prostate with cystitis, and in the urethra from stricture. It is stated that slight grades of hydronephrosis have been found in patients with excessive polyuria.

The cystoscope may be used for this purpose.

In whatever way produced, when the ureter is blocked the secretion accumulates in the pelvis and infundibula. Sometimes acute inflammation follows, but more commonly the slow, gradual pressure causes atrophy of the papillæ with gradual distention and wasting of the organ. In acquired cases from pressure, even when dilatation is extreme, there may usually be seen a thin layer of renal structure. In the most extreme stages the kidney is represented by a large cyst, which may perhaps show on its inner surface imperfect septa. The fluid is thin and yellowish in color, and contains traces of urinary salts, urea, uric acid, and sometimes albumen. The secretion may be turbid from admixture with small quantities of pus.

Total occlusion does not always lead to a hydronephrosis, but may be followed by atrophy of the kidney. It appears that when the obstruction is intermittent or not complete the greatest dilatation is apt to follow. The sac may be enormous, and cause an abdominal tumor of the largest size. The condition has even been mistaken for ascites. Enlargement of the other kidney may compensate for the defect. Hypertrophy of the left side of the heart usually follows.

Symptoms.—When small, it may not be noticed. The congenital cases when bilateral usually prove fatal within a few days; when unilateral, the tumor may not be noticed for some time. It increases progressively and has all the characters of a tumor in the renal region. In adult life many of the cases, due to pressure by tumors, as in cancer of the uterus and enlargement of the prostate, etc., give rise to no symptoms.

There are remarkable instances of *intermittent* hydronephrosis in which the tumor suddenly disappears with the discharge of a large quantity of clear fluid. The sac gradually refills, and the process may be repeated for years. In these cases the obstruction is unilateral; a cicatricial stricture exists, or a valve is present in the ureter, or the ureter enters the upper part of the pelvis.

The examination of the abdomen shows, in unilateral hydronephrosis, a tumor occupying the renal region. When of moderate size it is readily recognized, but when large it may be confounded with ovarian or other tumors. In young children it may be mistaken for sarcoma of the kidney or of the retroperitoneal glands, the common causes of abdominal tumor in early life. Aspiration alone would enable us to differentiate between hydronephrosis and tumor. The large hydronephrotic sac is frequently mistaken for ovarian tumor. The latter is, as a rule, more mobile, and rarely fills the deeper portion of the lumbar region so thoroughly. The ascending colon can often be detected passing over the renal tumor, and examination per vaginam, particularly under ether, will give important indications as to the condition of the ovaries. In doubtful cases the sac should be aspirated. The fluid of the renal cyst is clear, or turbid from the presence of cell elements, rarely colloid in character; the specific gravity is low; albumen and traces of urea and uric acid are usually present; and the epithelial elements in it may be similar to those found in the pel-

vis of the kidney. In old sacs, however, the fluid may not be characteristic, since the urinary salts disappear, but in one case of several years' duration oxalate of lime and urea were found.

Perhaps the greatest difficulty is offered by the condition of hydronephrosis in a movable kidney. Here, the history of sudden disappearance of the tumor with the passage of a large quantity of clear fluid would be a point of great importance in the diagnosis. In those rare instances of an enormous sac filling the entire abdomen, and sometimes mistaken for ascites, the character of the fluid might be the only point of difference. The tumor of pyonephrosis may be practically the same in physical characteristics. Fever is usually present, and pus is often found in the urine. In these cases, when in doubt, exploratory puncture should be made.

The outlook in hydronephrosis depends much upon the cause. When single, the condition may never produce serious trouble, and the intermittent cases may persist for years. The latter are the most hopeful, and Frederick Taylor mentions an instance in which, after the fifth or sixth subsidence, in the course of two years, a calculus was discharged. Occasionally the cyst ruptures into the peritonæum, more rarely through the diaphragm into the lung. A remarkable case of this kind is at present under the care of my colleague, Halsted. A man, aged twenty-one, had, from his second year, attacks of abdominal pain in which a swelling would appear between the hip and costal margin and subside with the passage of a large amount of urine. In January, 1888, the sac discharged through the right lung.* Reaccumulations have occurred on several occasions since, and on June 9, 1891, the sac was opened and drained.

The sac may discharge spontaneously through the ureter and the fluid never reaccumulate. In bilateral hydronephrosis there is a danger that uræmia may supervene. There are instances, too, in which blocking of the ureter on the sound side by calculus has been followed by uræmia. And, lastly, the sac may suppurate, and the condition change to one of pyonephrosis.

Treatment.—Cases of intermittent hydronephrosis which do not cause serious symptoms should be let alone. It is stated that, in sacs of moderate size, the obstruction has been overcome by shampooing. If practised, it should be done with great care. When the sac reaches a large size aspiration may be performed and repeated if necessary. Puncture should be made in the flank, midway between the ilium and the last rib. If the fluid reaccumulates and the sac becomes large, it may be incised and drained, or, as a last resort, the kidney may be removed.

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* Sowers, New York Medical Record, 1888.

X. NEPHROLITHIASIS (*Renal Calculus*).

Definition.—The formation in the kidney or in its pelvis of concretions, by the deposition of certain of the solid constituents of the urine.

Etiology and Pathology.—In the kidney substance itself the separation of the urinary salts produces a condition to which, unfortunately, the term infarct has been applied. Three varieties may be recognized: (1) The uric-acid infarct, usually met with at the apices of the pyramids in new-born children and during the first weeks of life. It is readily recognized as a yellowish linear streak in the pyramids and is of no significance; (2) the urate of soda infarct, sometimes associated with urate of ammonia, which forms whitish lines at the apices of the pyramids and is met with chiefly, but not always, in gouty persons; and (3) the lime infarcts, forming very opaque white lines in the pyramids, usually in old people.

In the pelvis and calyces concretions of the following forms occur: (*a*) Small gritty particles, *renal sand*, ranging in size from the individual grains of the uric-acid sediment to bodies one or two millimetres in diameter. These may be passed in the urine for long periods without producing any symptoms, since they are too fine to be arrested in their downward passage.

(*b*) Larger concretions, ranging in size from a small pea to a bean, and either solitary or multiple in the calyces and pelvis. It is the smaller of these calculi which, in their passage, produce the attacks of renal colic. They may be rounded and smooth, or present numerous irregular projections.

(*c*) The dendritic form of calculus. The orifice of the ureter may be blocked by a Y-shaped stone. The pelvis itself may be occupied by the concretion, which forms a more or less distinct mould. These are the remarkable *coral calculi*, which form in the pelvis complete moulds of infundibula and calyces, the latter even presenting cup-like depressions corresponding to the apices of the papillæ. Some of these casts in stone of the renal pelvis are as beautifully moulded as Hyrtl's corrosion preparations.

Chemically the varieties of calculi are: (1) Uric acid, by far the most important, which may form the renal sand, the small solitary, or the large dendritic stones. They are very hard, the surface is smooth, and the color reddish. The larger stones are usually stratified and very dense. Usually the uric acid and the urates are mixed, but in children stones composed of urates alone may occur.

(2) Oxalate of lime, which forms mulberry-shaped calculi, studded with points and spines. They are often very dark in color, intensely hard, and are a mixture of oxalate of lime and uric acid.

(3) Phosphatic calculi are composed of the phosphate of lime and the ammonio-magnesium phosphate, sometimes mixed with a small amount of

carbonate of lime. They are not common, since the phosphatic salts are oftener deposited about the uric acid or the oxalate of lime stones.

(4) Rare forms of calculi are made up of cystine, xanthine, carbonate of lime, indigo, and urostealith.

The mode of formation of calculi has been much discussed. They may be produced by an excess of a sparingly soluble abnormal ingredient, such as cystine or xanthine; more frequently by the presence of uric acid in a very acid urine which favors its deposition. Sir William Roberts thus briefly states the conditions which lead to the formation of the uric-acid concretions: high acidity, poverty in salines, low pigmentation, and high percentage of uric acid. The presence of albumen and mucus may determine, as Ord suggests, the deposition of the uric acid and thus form the starting point of a stone. Ova of parasites, blood-clot, casts, and shreds of epithelium may form the nuclei of stones.

Renal calculi are most common in the early and later periods of life. They are moderately frequent in this country, but there do not appear to be special districts, corresponding to the "stone counties" in England. Men are more often affected than women. Sedentary occupations seem to predispose to stone.

The effects of the calculi are varied. It is by no means uncommon to find a dozen or more stones of various sizes in the calyces without any destruction of the mucous membrane or dilatation of the pelvis. A turbid urine fills the pelvis in which there are numerous cells from the epithelial lining. There are cases of this sort in which, apparently, the stones may go on forming and are passed for years without seriously impairing the health and without inconvenience, except the attacks of renal colic. Still more remarkable are the cases of coral-like calculi, which may occupy the entire pelvis and calyces without causing pyelitis, but which gradually lead to more or less induration of the kidney. The most serious effects are when the stone excites a suppurative pyelitis and pyonephrosis.

Symptoms.—Patients may pass gravel for years without having an attack of renal colic, and a stone may never lodge in the ureter. In other instances, the formation of calculi goes on year by year and the patient has recurring attacks such as have been so graphically described by Montaigne in his own case. A patient may pass an enormous number of calculi. Some years ago I was consulted by a commercial traveller, an extremely vigorous man, who for many years had repeated attacks of renal colic, and had passed several hundred calculi of various sizes. His collection filled an ounce bottle. A patient may pass a single calculus, and never be troubled again. The large coral calculi may excite no symptoms. In a remarkable specimen of the kind, presented to the McGill Medical Museum by J. A. Macdonald, the patient, a middle-aged woman, died suddenly with uræmic symptoms. There was no pyelitis, but the kidneys were sclerotic.

Renal colic ensues when a stone enters the ureter. An attack may set in abruptly without apparent cause, or may follow a strain in lifting. It is characterized by agonizing pain, which starts in the flank of the affected side, passes down the ureter, and is felt in the testicle and along the inner side of the thigh. The pain may also radiate through the abdomen and chest, and be very intense in the back. In severe attacks there are nausea and vomiting and the patient is collapsed. The perspiration breaks out upon the face and the pulse is feeble and quick. A chill may precede the outbreak, and the temperature may rise as high 103°. No one has more graphically described an attack of "the stone" than Montaigne,* who was a sufferer for many years: "Thou art seen to sweat with pain, to look pale and red, to tremble, to vomit well-nigh to blood, to suffer strange contortions and convulsions, by starts to let tears drop from thine eyes, to urine thick, black, and frightful water, or to have it suppressed by some sharp and craggy stone, that cruelly pricks and tears thee." The symptoms persist for a variable period. In short attacks they do not last longer than an hour; in other instances they continue for a day or more, with temporary relief. Micturition is frequent, occasionally painful, and the urine, as a rule, is bloody. There are instances in which a large amount of clear urine is passed, probably from the other kidney. In rare cases the secretion of urine is completely suppressed, even when the kidney on the opposite side is normal, and death may occur from uræmia. This most frequently happens when the second kidney is extensively diseased, or when only a single kidney exists. A number of cases of this kind have been recorded. The condition has been termed, by Sir William Roberts, obstructive suppression. It is met with also when cancer compresses both ureters or involves their orifices in the bladder. The patient may not appear to be seriously ill at first, and uræmic symptoms may not develop for a week, when twitching of the muscles, great restlessness, and sometimes drowsiness supervene, but, strange to say, neither convulsions nor coma. Death takes place usually within twelve days from the onset of the obstruction.

After the attack of colic has passed there is more or less aching on the affected side, and the patient can usually tell from which kidney the stone has come. Examination during the attack is usually negative. Very rarely the kidney becomes palpable. Tenderness on the affected side is common. In very thin persons it may be possible, on examination of the abdomen, to feel the stone in the ureter; or the patient may complain of a grating sensation.

When the calculi remain in the kidney they may produce very definite and characteristic symptoms, of which the following are the most important:

(1) *Pain*, usually in the back, which is often no more than a dull sore-

* Essays, Book III, 13.

ness, but which may be severe and come on in paroxysms. It is usually on the side affected, but may be referred to the opposite kidney, and there are instances in which the pain has been confined to the sound side. Pains of a similar nature may occur in movable kidneys, and there are several instances on record in which surgeons have incised the kidney for stone and found none.

(2) *Hæmaturia.*—Although this occurs most frequently when the stone becomes engaged in the ureter, it may also come on when the stones are in the pelvis. The bleeding is seldom profuse, as in cancer, but in some instances may persist for a long time. It is aggravated by exertion and lessened by rest. Frequently it only gives to the urine a smoky hue. The urine may be free for days, and then a sudden exertion or a prolonged ride may cause smokiness, or blood may be passed in considerable quantities.

(3) *Pyelitis.*—(*a*) There may be attacks of severe pain in the back, not amounting to actual colic, which are initiated by a heavy chill followed by fever, in which the temperature may reach 104° or 105°, followed by profuse sweating. The urine, which has been clear, may become turbid and smoky and contain blood and abundant epithelium from the pelvis. Attacks of this description may recur at intervals for months or even years, and are generally mistaken for malaria, unless special attention is paid to the urine and to the existence of the pain in the back. This renal intermittent fever, due to the presence of calculi, is identical with the hepatic intermittent fever, due to gall-stones, and in both it is important to remember that the most intense paroxysms may occur without any evidence of suppuration.

(*b*) More frequently the symptoms of purulent pyelitis, which have already been described, are present; pain in the renal region, recurring chills, and pus in the urine, with or without indications of pyonephrosis.

(4) *Pyuria.*—There are instances of stone in the kidney in which pus occurs continuously or intermittently in the urine for many years. On many occasions between 1875 and 1884 I examined the urine of a physician who had passed calculi when a student in 184[illegible], and has had pus in the urine at intervals ~~ever since. There was no tumor. He had never had a second attack of colic~~. In spite of the prolonged suppuration he has had remarkable mental and bodily vigor.

Patients with stone in the kidney are often robust, high livers, and gouty. Attacks of dyspepsia are not uncommon, or they may have severe headaches.

Diagnosis.—Renal may be mistaken for intestinal colic, particularly if the distention of the bowels is marked, or for biliary colic. The situation and direction of the pain, the retraction and tenderness of the testicle, the occurrence of hæmaturia, and the altered character of the urine are distinctive features. Attention may again be called to the fact that attacks simulating renal colic are associated with movable kidney, or even, it has been supposed, without mobility of the kidney, with the accumu-

Jean ~~D. G. Steele 2322 Fillmore St. Frisco~~
~~I know of one~~ instance, in which pain ~~has been~~ was present for a couple years in the right renal region
& a surgeon ~~made~~ an exploring incision & found only
a contracted Kidney

5
to 1891 when I last heard from him

lation of the oxalates or uric acid in the pelvis of the kidney. The diagnosis between a stone in the kidney and stone in the bladder is not always easy, though in the latter the pain is particularly about the neck of the bladder, and not limited to one side. Important points are the reaction of the urine, which in stone in the bladder is almost invariably alkaline, and the abundance of mucus with the pus. It is stated that certain differences occur in the symptoms produced by different sorts of calculi. The large uric-acid calculi less frequently produce severe symptoms. On the other hand, as the oxalate of lime is a rougher calculus, it is apt to produce more pain (often of a radiating character) than the lithic-acid form, and to cause hæmorrhage. In both these forms the urine is acid. The phosphatic calculi are stated to produce the most intense pain, and the urine is commonly alkaline.

Treatment.—In the attacks of renal colic great relief is experienced by the hot bath, which is sometimes sufficient to relax the spasm. When the pain is very intense morphia should be given hypodermically, and inhalations of chloroform may be necessary until the effects of the anodyne are manifest. Local applications are sometimes grateful—hot poultices, or cloths wrung out of hot water. The patient may drink freely of hot lemonade, soda water, or barley water. Occasionally change in posture will give great relief, and inversion of the patient is said to be followed by immediate cessation of the pain.

In the intervals the patient should, as far as possible, live a quiet life, avoiding sudden exertion of all sorts. The essential feature in the treatment is to keep the urine abundant and, in a majority of the cases, alkaline. The patient should drink daily a large but definite quantity of mineral waters * or distilled water, which is just as satisfactory. The citrate or bicarbonate of potash may be added. The aching pains in the back are often greatly relieved by this treatment. Many patients find benefit from a stay at Saratoga, Bedford, Poland, or other mineral springs in this country, or at Vichy or Ems in Europe.

The diet should be carefully regulated, and similar to that indicated in the early stages of gout. Sir William Roberts recommends what is known as the solvent treatment for uric-acid calculi. The citrate of potash is given in large doses of half a drachm to a drachm every three hours in a tumblerful of water. This should be kept up for several months. I have had no success with this treatment, nor, when one considers the character of the uric-acid stones usually met with in the kidney, does it seem likely that any solvent action could be exercised upon them by changes in the urine. This treatment should be abandoned if the urine becomes ammoniacal.

The surgical treatment of stone in the kidney has advanced rapidly in the hands of Morris and others. It should be resorted to only when the

* Some of these, if we judge by the laudatory reports, are as potent as the waters of Corsena, declared by Montaigne to be "powerful enough to break stones."

attacks of pain are of such frequency as to interfere with the occupation of the patient, or when pyelitis or pyelonephritis has been excited. Stone in the kidney is not inconsistent with a long life and with the enjoyment of a fair measure of health.

XI. TUMORS OF THE KIDNEY.

These are benign and malignant. Of the benign tumors, the most common are the small nodular *fibromata* which occur frequently in the pyramids, the *aberrant adrenals* which Grawitz has described, and occasionally *lipoma*, *angioma*, or *lymphadenoma*. The *adenomata* may be congenital. In one of my cases the kidneys were greatly enlarged, contained small cysts, and numerous adenomatous structures throughout both organs.

Malignant growths—*cancer* or *sarcoma*—may be either primary or secondary. The sarcomata are the most common, either alveolar sarcoma or the remarkable form containing striped muscular fibres—rhabdo-myoma. Carcinoma is less frequent, and is of the encephaloid variety.

Primary cancer—meaning by this, malignant disease—is not uncommon, and the statistics given by some writers do not represent the frequency with which it is met with, at any rate, in this country. Virchow gives the ratio to cancer in other parts as one half of one per cent.

The tumors attain a very large size. In one of my cases the left kidney weighed twelve pounds and almost filled the abdomen. In children they may reach an enormous size. Morris states that in a boy at the Middlesex Hospital the tumor weighed thirty-one pounds. They grow rapidly, are often soft, and hæmorrhage frequently takes place into them. In the sarcomata invasion of the pelvis or of the renal vein is common. The rhabdo-myomas rarely form very large tumors, and death occurs shortly after birth. In one of my cases the child lived to the age of three years and a half. The tumor grew into the renal vein and inferior cava. A detached fragment passed as an embolus into the pulmonary artery, and a portion of it blocked the tricuspid orifice.

Symptoms.—The following are the most important: (1) Hæmaturia. This may be the first indication. The blood is fluid or clotted, and there may be very characteristic moulds of the pelvis of the kidney and of the ureter. It would no doubt be possible for such to form in the hæmaturia from calculus, but I have never met with a case of blood-casts of the pelvis and of the ureter, either alone or together, except in cancer. It is rare indeed that cancer elements may be recognized as in the urine. Of the numerous specimens which I have examined, in not one have I found elements which could be clearly distinguished from the multiform transitional epithelium constantly present in these cases.

(2) Pain is an uncertain symptom. In several of the largest tumors

which have come under my observation there has been no discomfort from beginning to close. When present, it is of a dragging, dull character, situated in the flank and radiating down the thigh. The passage of the clots may cause great pain

(3) Progressive emaciation. The loss of flesh is usually marked and advances rapidly. There may, however, be a very large tumor without emaciation.

Physical Signs.—In almost all instances tumor is present. When small and on the right side, it may be very movable; in some instances, occupying a position in the iliac fossa, it has been mistaken for ovarian tumor. The large growths fill the flank and gradually extend toward the middle line, occupying the right or left half of the abdomen. Inspection may show two or three hemispherical projections corresponding to distended sections of the organ. In children the abdomen may reach an enormous size and the veins are prominent and distended. On bimanual palpation the tumor is felt to occupy the lumbar region and can usually be lifted slightly from its bed; in some cases it is very movable, even when large; in others it is fixed, firm, and solid. The respiratory movements have but slight influence upon it. Rapidly growing renal tumors are soft, and on palpation may give a sense of fluctuation. A point of considerable importance is the fact that the colon crosses the tumor, and can usually be detected without difficulty.

Diagnosis.—In children very large abdominal tumors are either renal or retroperitoneal. The retroperitoneal sarcoma (Lobstein's cancer) is more central, but may attain as large a size. If the case is seen only toward the end, a differential diagnosis may be impossible; but as a rule the sarcoma is less movable. It is to be remembered that these tumors may invade the kidney. On the left side an enlarged spleen is readily distinguished, as the edge is very distinct and the notch or notches well marked; it descends during respiration, and the colon lies behind, not in front of it. On the right side growths of the liver are occasionally confounded with renal tumors; but such instances are rare, and there can usually be detected a zone of resonance between the upper margin of the renal tumor and the ribs. Late in the disease, however, this is not possible, for the renal tumor is in close union with the liver.

A malignant growth in a movable kidney may be very deceptive and may simulate cancer of the ovary or fibroid of the uterus. The great mobility upward of the renal growth and the negative result of examination of the pelvic viscera are the reliable points.

Medicinal treatment is of no avail. When the growth is small and the patient in good condition removal of the organ may be undertaken, but the percentage of cases of recovery is very small.

XII. CYSTIC DISEASE OF THE KIDNEY.

The following varieties of cysts are met with:

(1) The small cysts, already described in connection with the chronic nephritis, which result from dilatation of obstructed tubules or of Bowman's capsules.

(2) Solitary cysts, ranging in size from a marble to an orange, or even larger, are occasionally found in kidneys which present no other changes. They never give rise to symptoms, though, in exceptional cases, they may form tumors of considerable size. They, too, in all probability, result from obstruction.

(3) The congenital cystic kidneys. In this remarkable condition the kidneys are represented by a conglomeration of cysts, varying in size from a pea to a marble. The organs are greatly enlarged, and together may weigh six or more pounds. In the fœtus they may attain a size sufficient to impede labor. Little or no renal tissue may be noticeable, although in microscopical sections it is seen that a considerable amount remains in the interspaces. The cysts contain a clear or turbid fluid, sometimes reddish brown or even blackish in color, and may be of a colloidal consistence. Albumen, blood crystals, cholesterin, with triple phosphates and fat drops are found in the contents. Urea and uric acid are rarely present. The cysts are lined by a flattened epithelium. It is not yet accurately known how these cysts originate. That it is a defect in development rather than a pathological change is suggested by the fact that it is often in the embryo associated with other anomalies, particularly imperforate anus. Both Shattock and Bland Sutton, who have studied the question carefully, believe that the anomaly of development is in the failure of complete differentiation of the Wolffian bodies, which are, as it were, mixed with the kidneys and give rise to the cysts.

In a large majority of the cases death occurs, either *in utero* or shortly after birth; but instances are met with at all ages up to fifty or sixty, and I see no reason to suppose that these are not instances of persistence of the congenital form.

In the adult the tumors may be felt in the lumbar region as large rounded masses.

The *symptoms* are those of chronic interstitial nephritis. Many of the cases have presented no indications whatever until a sudden attack of uræmia; others have died of heart-failure. A rare termination in a case at the University Hospital, Philadelphia, was the rupture of one of the cysts and the production of a perinephritic abscess. The cardio-vascular changes induced are similar to those of interstitial nephritis. The left ventricle is hypertrophied and the arterial tension is greatly increased. The condition is compatible with excellent health. The dangers are those associated with chronic Bright's disease. It is important to remember that the conglomerate cystic kidney is almost invariably bilat-

all right

There are cases very difficult to classify, in which the kidneys are greatly enlarged, & very cystic in middle aged or elderly persons, & yet not so large as the congenital form (see Path. Jl. H. in case of mine Oct 13th 1893

Though the condition is congenital - yet from the history of certain cases - it is evident that the organs must increase enormously in size - thus in a patient of Dr. Alfred King's of Portland - Me - a man aged 54. The abdomen presented nothing abnormal on careful examination three years before his death - but three months prior to this date there were large bilateral tumors in the renal region - which were readily diagnosed as cystic kidneys. The organs weighed four pounds each.

Make a section on cysts of the liver - Caspar Sherbler has met with a cyst lined by ciliated epithelium. (Deller Oct. 1893) in a case of death from abt aneurism

eral. One kidney may be somewhat larger and more cystic than the other.

The diagnosis can sometimes be made. Great enlargement of both organs, with hypertrophy of the left heart and increased arterial tension, would suggest the condition.

Operative interference is not justifiable. I know an instance in which one kidney was removed and the patient died within twenty-four hours.

(4) Occasionally the kidneys and liver present numerous small cysts scattered through the substance. The spleen also may be involved. The cysts in the kidney are small, and neither so numerous nor so thickly set as in the conglomerate form, though in these cases the condition is probably the result of some congenital defect. There are cases, however, in which the kidneys are very large. It is more common in the lower animals than in man. I have seen several instances of it in the hog; in one case the liver weighed forty pounds, and was converted into a mass of simple cysts. The kidneys were less involved. Charles Kennedy * states that he has found references to twelve cases of combined cystic disease of the liver and kidneys.

The echinococcus cysts will be spoken of under the section on parasites.

XIII. PERINEPHRIC ABSCESS.

Suppuration in the connective tissue about the kidney may follow (1) blows and injuries; (2) the extension of inflammation from the pelvis of the kidney, the kidney itself, or the ureters; (3) perforation of the bowel, most commonly the appendix, in some instances the colon; (4) extension of suppuration from the spine, as in caries, or from the pleura, as in empyema; (5) as a sequel of the fevers, particularly in children.

In the post-mortem examination of a case of perinephric abscess the kidney is found surrounded by pus, particularly at the posterior part, though the pus may lie altogether in front, between the kidney and the peritonæum. Usually the abscess cavity is large and extensive. The pus is often offensive and may have a distinctly fæcal odor from contact with the large bowel. It may burrow in various directions and may burst into the pleura and be discharged through the lungs. A more frequent direction is down the psoas muscle, when it appears in the groin, or it may pass along the iliacus fascia and appear at Poupart's ligament. It may perforate the bowel or rupture into the peritonæum, and in some instances it has penetrated the bladder or vagina.

Post mortem we occasionally find a condition of chronic perinephritis in which the fatty capsule of the kidney is extremely firm, with numer-

* Laboratory Reports of the Royal College of Physicians, Edinburgh, vol. iii.

ous bands of fibrous tissue, and is stripped off from the proper capsule with the greatest difficulty. Such a condition probably produces no symptoms.

Symptoms.—There may be intense pain, aggravated by pressure, in the lumbar region. In other instances, the onset is insidious; there is no pain in the renal region, but on the first examination signs of deep-seated suppuration may be detected. On the affected side there is usually pain, which may be referred to the neighborhood of the hip-joint or radiate down the thigh and be associated with retraction of the testis. Sometimes the pain is referred even to the knee-joint, as in hip-disease. The patient lies with the thigh flexed, so as to relax the psoas muscle, and in walking throws, as far as possible, the weight on the opposite leg. According to Gibney, the patient keeps the spine immobile, assumes a stooping posture in walking, and has great difficulty in voluntarily adducting the thigh.

There may be pus in the urine if the disease has extended from the pelvis or the kidney, but in other forms the urine is clear. When pus has formed there are usually chills with irregular fever and sweats. On examination, deep-seated induration is felt between the last rib and the crest of the ilium. Bimanual palpation may reveal a distinct tumor mass. Œdema or puffiness of the skin is frequently present.

The diagnosis of perinephric abscess is usually easy, and in any case when doubt exists the aspirator needle should be used. We cannot always differentiate the primary forms from those due to perforation of the kidney or of the bowel. This, however, makes but little difference, for the treatment is identical. It is usually possible by the history and examination to exclude disease of the vertebra. In children the condition is often mistaken for disease of the hip-joint, but the pain is higher, and there is an entire absence of fulness and tenderness over the hip-joint itself.

From whatever cause produced, the indications for treatment are identical—early, free, and permanent drainage.

SECTION VIII.

DISEASES OF THE NERVOUS SYSTEM.

I. DISEASES OF THE NERVES.

1. NEURITIS (*Inflammation of the Nerve Fibres*).

Neuritis may be *localized* in a single nerve, or *general*, involving a large number of nerves, in which case it is usually known as *multiple neuritis* or *polyneuritis*.

Etiology.—*Localized neuritis* arises from (*a*) cold, which is a very frequent cause, as, for example, in the facial nerve. This is sometimes known as rheumatic neuritis. (*b*) Traumatism—wounds, blows, direct pressure on the nerves, the tearing and stretching which follow a dislocation or a fracture, and the hypodermic injection of ether. Under this section come also the professional palsies, due to pressure in the exercise of certain occupations. (*c*) Extension of inflammation from neighboring parts, as in a neuritis of the facial nerve due to caries in the temporal bone, or in that met with in syphilitic disease of the bones, disease of the joints, and occasionally in tumors.

Multiple neuritis has a very complex etiology, the causes of which may be classified as follows: (*a*) The poisons of infectious diseases, as in leprosy, diphtheria, typhoid fever, small-pox, scarlet fever, and occasionally in other forms; (*b*) the organic poisons, comprising the diffusible stimulants, such as alcohol and ether, bisulphide of carbon, and naphtha, and the metallic bodies, such as lead, arsenic, and mercury; (*c*) cachectic conditions, such as occur in anæmia, cancer, tuberculosis, or marasmus from any cause; (*d*) the endemic neuritis or beri-beri; and (*e*) lastly, there are cases in which none of these factors prevail, but the disease sets in suddenly after overexertion or exposure to cold.

Morbid Anatomy.—In neuritis due to the extension of inflammation the nerve is usually swollen, infiltrated, and red in color. The inflammation may be chiefly perineural or it may pass into the deeper portion—*interstitial* neuritis—in which form there is an accumulation of lymphoid elements between the nerve bundles. The nerve fibres themselves may not appear involved, but there is an increase in the nuclei of

the sheath of Schwann. The myelin is fragmented, the nuclei of the internodal cells are swollen, and the axis cylinders present varicosities or undergo granular degeneration. Ultimately the nerve fibres may be completely destroyed and replaced by a fibrous connective tissue in which much fat is sometimes deposited—the *lipomatous neuritis* of Leyden.

In other instances the condition is termed *parenchymatous* neuritis, in which the changes are like those met with in the secondary or Wallerian degeneration, which follows when the nerve is cut off from its centre. The medullary substance and the axis cylinders are chiefly involved, the interstitial tissue being but little altered or only affected secondarily. The myelin becomes segmented and divides into small globules and granules, and the axis cylinders become granular, broken, subdivided, and ultimately disappear. The nuclei of the sheath of Schwann proliferate and ultimately the fibres are reduced to a state of atrophic tubes without a trace of the normal structure. The muscles connected with the degenerated nerves usually show marked atrophic changes, and in some instances the change in the nerve sheath appears to extend directly to the interstitial tissue of the muscles—the *neuritis fascians* of Eichhorst.

Symptoms. (*a*) **Localized Neuritis.**—As a rule the constitutional disturbances are slight. The most important symptom is pain of a boring or stabbing character, usually felt in the course of the nerve and in the parts to which it is distributed. The nerve itself is sensitive to pressure, probably, as Weir Mitchell suggests, owing to the irritation of its nervi nervorum. The skin may be slightly reddened or even œdematous over the seat of the inflammation. Mitchell has described increase in the temperature and sweating in the affected region, and such trophic disturbances as effusion into the joints and herpes. The function of the muscle to which the nerve fibres are distributed is impaired, motion is painful, and there may be twitchings or contractions. The tactile sensation of the part may be somewhat deadened, even when the pain is greatly increased. In the more chronic cases of local neuritis, such, for instance, as follow the dislocation of the humerus, the localized pain, which at first may be severe, gradually disappears, though some sensitiveness of the brachial plexus may persist for a long time, and the nerve cords may be felt to be swollen and firm. The pain is variable—sometimes intense and distressing; at others not causing much inconvenience. Numbness and formication may be present and the tactile sensation may be greatly impaired. The motor disturbances are marked. Ultimately there is extreme atrophy of the muscles. Contractures may occur in the fingers. The skin may be reddened or glossy, the subcutaneous tissue œdematous, and the nutrition of the nails may be defective.

A neuritis limited at first to a peripheral nerve may extend upward—the so-called ascending or migrating neuritis—and involve the larger nerve trunks, or even reach the spinal cord, causing subacute myelitis (Gowers). Thus, in a case reported by James Stewart, a girl of fourteen

had severe pain in the big toe of the left foot, which gradually extended up the leg and resisted all treatment until a portion of the sciatic nerve was removed. A year later she had pain in the little finger of the left hand, which gradually ascended along the ulnar nerve and required for its relief division and stretching. It has been suggested that the paralysis secondary to visceral disease, as of the bladder, may be due to an ascending neuritis. The inflammation may extend to the nerves of the other side, either through the spinal cord or its membranes, or without any involvement of the nerve centres, the so-called sympathetic neuritis. The electrical changes in localized neuritis vary a great deal, depending upon the extent to which the nerve is injured. The lesion may be so slight that the nerve and the muscles to which it is distributed may react normally to both currents; or it may be so severe that the typical reaction of degeneration develops within a few days, i. e., the nerve does not respond to stimulation by either current while the muscle reacts only to the galvanic current and in a peculiar manner. The contraction caused is slow and lazy, instead of sharp and quick as in the normal muscle, and the AnC contraction is usually stronger than the CC contraction. Between these two extremes there are many different grades and a careful electrical examination is most important as an aid to diagnosis and prognosis.*

The duration varies from a few days to weeks or months. A slight traumatic neuritis may pass off in a day or two, while the severer cases, such as follow unreduced dislocation of the humerus, may persist for months or never be completely relieved.

(*b*) **Multiple Neuritis.**—This presents a complex symptomatology. The following are the most important groups of cases:

(1) *Acute Febrile Polyneuritis.*—The attack follows exposure to cold or overexertion, or, in some instances, comes on spontaneously. The onset resembles that of an acute infectious disease. There may be a definite chill, pains in the back and limbs or joints, so that the case may be thought to be acute rheumatism. The temperature rises rapidly and may reach 103° or 104°. There are headache, loss of appetite, and the general symptoms of acute infection. The limbs and back ache. Intense pain in the nerves, however, is by no means constant. Tingling and formication are felt in the fingers and toes, and there is increased sensitiveness of the nerve trunks or of the entire limb. Loss of muscular power, first marked, perhaps, in the legs, gradually comes on and extends with the features of an ascending paralysis. In other cases the paralysis begins in the arms. The extensors of the wrists and the flexors of the ankles are early affected, so that there is foot and wrist drop. In severe cases there is general loss of muscular power, producing a flabby paralysis, which may extend to the muscles of the face and to the intercostals, and respiration may be carried on by the diaphragm alone. The muscles soften and waste rapidly. There

* See under facial paralysis.

may be only hyperæsthesia with soreness and stiffness of the limbs; in some cases, increased sensitiveness with anæsthesia; in other instances the sensory disturbances are slight. The clinical picture is not to be distinguished, in many cases, from Landry's paralysis; in others, from the subacute myelitis of Duchenne. James Ross concludes from an analysis of all the reported cases of the former disease that it coincides with multiple neuritis in general etiology, symptoms, and course. On the other hand, Hun, in a very thorough study of a recent case of Landry's paralysis, concludes that it is a separate and distinctive disease.

The course is variable. In the most intense forms the patient may die in a week or ten days, with involvement of the respiratory muscles or from paralysis of the heart. As a rule in cases of moderate severity, after persisting for five or six weeks, the condition remains stationary and then slow improvement begins. The paralysis in some muscles may persist for many months and contractures may occur from shortening of the muscles, but even when this occurs the outlook is, as a rule, good, although the paralysis may have lasted for a year or more.

(2) *Recurring Multiple Neuritis.*—Under the term *polyneuritis recurrens* Mary Sherwood has described from Eichhorst's clinic two cases in adults—in one case involving the nerves of the right arm, in the other both legs. In one patient there were three attacks, in the other two, the distribution in the various attacks being identical. There has recently been at my clinic a somewhat similar case—a man, aged thirty-one, who had, two and a half years ago, widespread paralysis, and who now has a second attack.

(3) *Alcoholic Neuritis.*—This, perhaps the most important form of multiple neuritis, was described in 1822 by James Jackson, Sr., of Boston, whose account of it is very graphic. Wilks recognized it as alcoholic paraplegia, but the starting point of the recent researches on the disease dates from the observation of Dumenil, of Rouen. Of late years our knowledge of the disease has extended rapidly, owing to the researches of Huss, Leyden, James Ross, Buzzard, and Henry Hun. It occurs most frequently in women, particularly steady, quiet tipplers. Its appearance may be the first revelation to the physician or to the family of habits of secret drinking. The onset is usually gradual, and may be preceded for weeks or months by neuralgic pains and tingling in the feet and hands. Convulsions are not uncommon. Fever is rare. The paralysis gradually sets in, at first in the feet and legs, and then in the hands and forearms. The extensors are affected more than the flexors, so that there is wrist-drop and foot-drop. The paralysis may be thus limited and not extend higher in the limbs. In other instances there is paraplegia alone, while in the most extreme cases all the extremities are involved. In rare instances the facial muscles and the sphincters are also affected. A case with this distribution recovered in my wards last year. The sensory symptoms are very variable. There are cases in which there are numbness and tingling only, without

great pain. In other cases there are severe burning or boring pains, the nerve trunks are sensitive, and the muscles are sore when grasped. The hands and feet are frequently swollen and congested, particularly when held down for a few moments. The cutaneous reflexes as a rule are preserved. The deep reflexes are usually lost.

The course of these alcoholic cases is, as a rule, favorable, and after persisting for weeks or months improvement gradually begins, the muscles regain their power, and even in the most desperate cases recovery may follow. The extensors of the feet may remain paralyzed for some time, and give to the patient a distinctive walk, the so-called *steppage* gait, characteristic of peripheral neuritis. It is sometimes known as the pseudo-tabetic gait, although in reality it could not well be mistaken for the gait of ataxia. The foot is thrown forcibly forward, the toe lifted high in the air so as not to trip upon it. The heel is brought down first and then the entire foot. It is an awkward, clumsy gait, and gives the patient the appearance of constantly stepping over obstacles. Among the most striking features of alcoholic neuritis are the mental symptoms. Delirium is common, and hallucinations with extravagant ideas, resembling somewhat those of general paralysis. In some cases the picture is that of ordinary delirium tremens, but the most peculiar and almost characteristic mental disorder is that so well described by Wilks, in which the patient loses all appreciation of time and place, and describes with circumstantial details long journeys which he has recently taken, or tells of persons whom he has just seen.

(4) *Multiple Neuritis in the Infectious Diseases.*—There have been already referred to, particularly in diphtheria, in which it is most common. The peripheral nature of the lesion in these instances has been shown by post-mortem examination. The outlook is usually favorable and, except in diphtheria, fatal cases are uncommon. Multiple neuritis in tuberculosis, diabetes, and syphilis is of the same nature, probably due to toxic materials absorbed into the blood.

(5) *Arsenical and Saturnine Neuritis.*—The arsenical neuritis is not common; only a single instance of it has come under my observation. No case to my knowledge has followed the use of Fowler's solution in my ward or dispensary practice, although I am in the habit of giving in chorea and anæmia doses which might be regarded as excessive. The most common causes are accidental poisoning, as in the cases reported by Mills. In a case of E. G. Cutler the patient got the arsenic from green-paper tags, which he was in the habit of putting in his mouth. The general symptoms are not unlike those of alcoholic paralysis; the weakness of the extensors is marked and the *steppage* gait characteristic. The neuritis due to lead will be discussed in the consideration of lead poisoning. The special involvement of the motor nerves and the great frequency of the occurrence of wrist-drop are the peculiarities of this form.

A similar form of neuritis is caused by the bisulphide of carbon.

(6) *Endemic Neuritis; Beri-beri.*—This is a widely spread disease in parts of India, and in China and Japan. To Sheube and Baelz are due the credit of determining its true nature. It is probably due to a micro-organism. Food appears also to have a large share in its causation and it has been attributed to a fish diet. Some have thought it might be due to the presence of parasites in the intestines, but there are no grounds for this belief. There are several types of cases. In the acute pernicious form the nervous phenomena are not so marked. There are fever, anæmia, and general anasarca. In another group of cases there are numbness, loss of tendon reflexes, areas of anæsthesia, and muscular atrophy and anasarca. In other cases the paralysis and atrophy are the most prominent symptoms and the clinical picture is that of a rapidly progressing multiple neuritis with sensory and motor disturbances. The mortality varies from three or four to fifty per cent. Great difference of opinion still prevails concerning the cause of the disease. Special interest has been aroused in the subject in this country, owing to the fact that J. J. Putnam has described a similar disorder among the New England fishermen who frequent the Grand Banks. It occurs in epidemic form, and has, as prominent symptoms, general œdema, shortness of breath, and sensory disturbances with paralysis. In other instances, the paralysis is more extensive and proves fatal. In 1881 and 1889 there were epidemics among the crews of vessels fishing in this region. Birge describes eleven cases which occurred on one vessel in a crew of thirteen, two of whom died. One patient of this crew I saw with F. C. Shattuck, in the Massachusetts General Hospital, with the well-marked symptoms of multiple neuritis. The disease also exists in the West Indies, whence cases have come to this country (Seguin).

Diagnosis.—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion.

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of

all right

Overcrowding is has been a very important factor, particularly in ships.

Pe Metharny and Moulder

In this country the disease has been met with in the fishermen on the Newfoundland Banks. (J.J. Putnam)

It is not infrequently brought in the crews here from the West Indies & the East.

In Gt Britain an outbreak occurred last year (1894) in the Richmond Asylum Dublin in which 149 inmates were attacked [illegible] 17 died

the muscle and nerve are therefore important symptoms of multiple neuritis." *

There is rarely any difficulty in distinguishing the alcohol cases. The combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, is quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning or has diabetes.

Treatment.—Rest in bed is essential. In the acute cases with fever, the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the attendant must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit-drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sore an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often, with the most extreme deformity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

II. NEUROMATA.

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true

* Lectures on Neuritis, Medical Record, New York, 1887.

neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances, as in the case which I reported,* the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The true fascicular neuroma occurs in the form of the small subcutaneous painful tumor—*tubercula dolorosa*—which is situated on the nerves of the skin about the joints, sometimes on the face or on the breast. It is not always made up of nerve fibres, but may be, as shown by Hoggan, an adenomatous growth of the sweat glands.

The true neuromata, as a rule, are not painful, and occasionally are found associated with the nerve fibres in various regions. Those which develop at the ends and along the course of the nerves of the stump after amputation consist of connective tissue and of medullated and non-medullated nerve fibres. The most remarkable form is the *plexiform neuroma*, in which the various nerve cords are occupied by many hundreds of tumors. The cases are usually congenital. The tumors occur in all the nerves of the body. One of the most remarkable is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. Prudden † has collected forty-one cases from the literature, in a majority of which the peripheral nerves were affected.

Neuromata rarely cause symptoms, except the subcutaneous painful tumor or those in the amputation stump. Here they may be very painful and cause great distress. Motor symptoms are sometimes present, particularly a constant twitching. Epilepsy has sometimes been associated, and relief has followed removal of the growths.

The only available treatment is excision. The subcutaneous painful tumor does not return, and excision completely relieves the symptoms. On the other hand, the amputation neuromata may recur.

III. DISEASES OF THE CRANIAL NERVES.

I. Olfactory Nerve.

The functions of this nerve may be disturbed at its peripheral ending, at the bulb, in the course of the nerve, or at the central origin in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

(*a*) *Subjective Sensations; Parosmia.*—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an

* Journal of Anatomy and Physiology, vol. xv.

† American Journal of the Medical Sciences, vol. lxxx.

unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampal lobules. In rare instances, after injury of the head the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

(*b*) *Increased sensitiveness*, or *hyperosmia*, occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

(*c*) *Anosmia; Loss of the Sense of Smell.*—This may be produced by: (1) Affections of the termination of the nerve in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(2) The lesions of the bulb or of the nerves. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the nerve trunks may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, due possibly to atrophy of the nerves.

(3) Lesions of the olfactory centre. There are congenital cases in which the nerve structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which this symptom has been associated with disease in the hemisphere. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopical examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

II. Optic Nerve and Tract.

(1) *Lesions of the Retina.*

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye-

grounds. Only a brief reference can here be made to the more important of the appearances.

(*a*) **Retinitis.**—This occurs in certain general affections, more particularly in Bright's disease, syphilis, leukæmia, and anæmia. The common feature in all these states is the occurrence of hæmorrhage and the development of opacities. There may also be a diffuse cloudiness due to effusion of serum. The hæmorrhages are in the layer of nerve fibres. They vary greatly in size and form, but often follow the course of vessels. When recent the color is bright red, but they gradually change and old hæmorrhages are almost black. The white spots are due either to fibrinous exudate or to fatty degeneration of the retinal elements, and occasionally to accumulation of leucocytes or to a localized sclerosis of the retinal elements. The more important of the forms of retinitis to be recognized are:

Albuminuric retinitis, which occurs in chronic nephritis, particularly in the interstitial or contracted form. The percentage of cases affected is from fifteen to twenty-five. There are instances in which these retinal changes are associated with the granular kidney at a stage when the amount of albumen may be slight or transient; but in all such instances it will be found that there is a marked arterio-sclerosis. Gowers recognizes a degenerative form (most common), in which, with the retinal changes, there may be scarcely any alteration in the disk; a hæmorrhagic form, with many hæmorrhages and but slight signs of inflammation; and an inflammatory form, in which there is much swelling of the retina and obscuration of the disk. It is noteworthy that in some instances the inflammation of the optic nerve predominates over the other retinal changes and one may be in doubt for a time whether the condition is really associated with the renal changes or dependent upon intracranial disease.

Syphilitic Retinitis.—In the acquired form this is less common than choroiditis. In inherited syphilis *retinitis pigmentosa* is sometimes met with.

Retinitis in Anæmia.—It has long been known that a patient may become blind after a large hæmorrhage, either suddenly or within two or three days, and in one or both eyes. Occasionally the loss may be permanent and complete. In some of these instances a neuro-retinitis has been found, probably sufficient to account for the symptoms. In the more chronic anæmias, particularly in the pernicious form, retinitis is common, as determined first by Quincke.

In *malaria* retinitis or neuro-retinitis may be present, as noted by Stephen Mackenzie. It is seen only in the chronic cases with anæmia, and in my experience is not nearly so common proportionately as in pernicious anæmia. Of many instances which have come under my observation of severe malarial anæmia, particularly at the Philadelphia Hospital, there were only two with retinal hæmorrhages.

Leukæmic Retinitis.—In this affection the retinal veins are large

It may occur in epidemic form. (see Lancet. 1894. I. p. ii

and distended; there is also a peculiar retinitis, as described by Liebreich. It is not very common. Of the seventeen cases of leukæmia which have come under my observation, retinitis existed in only three of the ten in which the eye-grounds were examined. There are numerous hæmorrhages and white or yellow areas, which may be large and prominent. In one of my cases the retina post mortem was occupied by many small, opaque, white spots, looking like little tumors, the larger of which had a diameter of nearly two millimetres. In Case 13 of my series the leukæmia was diagnosed by Norris and De Schweinitz, at whose clinic the patient had applied on account of failing vision, from the condition of the eye-grounds alone.

Retinitis is also found occasionally in diabetes, in purpura, in chronic lead poisoning, and sometimes as an idiopathic affection.

(*b*) **Functional Disturbances of the Retina.** (1) *Toxic Amaurosis.*—This occurs in uræmia and may follow convulsions or come on independently. Thus, a patient who had become suddenly blind the previous day, was led into one of my wards at the Montreal General Hospital. He had had no special symptoms, but examination showed extensive cardio-vascular changes. The urine was albuminous. The ophthalmoscopic examination was negative. The condition, as a rule, persists only for a day or two. This form of amaurosis occurs in poisoning by lead and occasionally by quinine. It seems more probable that the poisons act on the centres and not on the retina.

(2) *Hysterical Amaurosis.*—More frequently this is loss of acuteness of vision—amblyopia—but the loss of sight in one or both eyes may apparently be complete. The condition will be mentioned subsequently under hysteria.

(3) *Tobacco Amblyopia.*—The loss of sight is usually gradual, equal in both eyes, and affects particularly the centre of the field of vision. The eye-grounds may be normal, but occasionally there is congestion of the disks. On testing the color fields a central scotoma for red and green is found in all cases. Ultimately, if the use of tobacco is continued, organic changes may develop with atrophy of the disk.

(4) *Night-blindness—nyctalopia*—the condition in which objects are clearly seen during the day or by strong artificial light, but become invisible in the shade or in twilight, and *hemeralopia*, in which objects cannot be clearly seen without distress in daylight or in a strong artificial light, but are readily seen in a deep shade or in twilight, are functional anomalies of the retina which rarely come under the notice of the physician.

(5) *Retinal hyperæsthesia* is sometimes seen in hysterical women, but is not found frequently in actual retinitis. I have seen it once, however, in albuminuric retinitis and once, in a marked degree, in a patient with aortic insufficiency, in whose retinæ there were no signs other than the throbbing arteries.

(2) *Lesions of the Optic Nerve.*

(*a*) **Optic Neuritis** (*Papillitis*; *Choked Disk*).—In the first stage there is congestion of the disk and the edges are blurred and striated. In the second stage, the congestion is more marked, the swelling increases, the striation also is more visible. The physiological cupping disappears and hæmorrhages are not uncommon. The arteries present little change, the veins are dilated, and the disk may swell greatly. In slight grades of inflammation the swelling gradually subsides and occasionally the nerve recovers completely. In instances in which the swelling and exudate are very great, the subsidence is slow, and when it finally disappears there is complete atrophy of the nerve. The retina not infrequently participates in the inflammation, which is then a neuro-retinitis.

This condition is of the greatest importance in diagnosis. It may exist in its early stages without any disturbance of vision, and even with extensive papillitis the sight may for a time be good.

Optic neuritis is seen occasionally in anæmia and lead poisoning, more commonly in Bright's disease as neuro-retinitis. It occurs occasionally as a primary idiopathic affection. The frequent connection with intracranial disease, particularly tumor, makes its presence of great value to practitioners. The nature of the growth is without influence. In over ninety per cent of such instances the papillitis is bilateral. It is also found in meningitis, either the tuberculous or the simple form. In meningitis it is easy to see how the inflammation may extend down the nerve sheaths. In the case of tumor it was thought at first that a choked disk resulted from increased pressure within the skull. It is now more commonly regarded, however, as a descending neuritis.

(*b*) **Optic Atrophy.**—This may be: (1) A primary affection. ~~Some of the cases have been hereditary and have come on~~ in all the males of a family shortly after puberty. A large number of the cases of primary atrophy are associated with spinal disease, particularly locomotor ataxia. Other causes which have been assigned for the primary atrophy are cold, sexual excesses, diabetes, the specific fevers, alcohol, and lead.

(2) Secondary atrophy results from cortical lesions, pressure on the chiasma or on the nerves, or, most commonly of all, is a sequence of papillitis.

The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former, the disk has a gray tint, the edges are well defined, and the arteries look almost normal; whereas in the consecutive atrophy the disk has a staring opaque-white aspect, with irregular outlines, and the arteries are very small.

The symptom of optic atrophy is loss of sight, proportionate to the damage in the nerve. The change is in three directions: "(1) Diminished acuity of vision; (2) alteration in the field of vision; and (3) altered perception of color." (Gowers.) The outlook in primary atrophy is bad; the

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majority of cases go on to complete blindness. In the consecutive form there is greater chance of retention of slight vision.

(3) *Affections of the Chiasma and Tract.*

At the chiasma the optic nerves undergo partial decussation. Each optic tract, as it joins the chiasma, contains nerve fibres which supply half of the retina of either eye. Thus, of the fibres of the right tract, part pass the chiasma without decussating and supply the temporal half of the right retina, the other and larger portion of the fibres of the tract decussate in the chiasma and join the left optic nerve, supplying the nasal half of the retina on the other side. The fibres which cross are in the middle portion of the chiasma, while the direct fibres are on each side. The following are the most important changes which ensue in lesions of the tract and of the chiasma:

(*a*) *Unilateral Affection of Tract* (Fig. 1 *B*).—If right this produces loss of function in the temporal half of the retina on the right side, and on the nasal half of the retina on the left side, so that there is only half vision, and the patient is blind to objects on the left side. This is termed homonymous hemianopia or lateral hemianopia. The fibres passing to the right half of each retina being involved, necessarily the left half of each visual field is blind. The hemianopia may be partial and only a portion of the half field may be lost. The affected visual fields may have the normal extent, but in some instances there is considerable reduction. ~~The color-vision is, as a rule, lost in the half field—hemiachromatopia—but the~~ half vision for color may be lost in central disease ~~without any change~~ in the field for white. When the left half of one field and the right half of the other, or vice versa, are blind, the condition is known as heteronymous hemianopia.

(*b*) *Disease of the Chiasma.*—(1) A lesion involves, as a rule, chiefly the central portion, in which the decussating fibres pass which supply the inner or nasal halves of the retinæ, producing in consequence loss of vision in the outer half of each field, or what is known as temporal hemianopia (Fig. 1 *H*).

(2) If the lesion is more extensive it may involve not only the central portion, but also the direct fibres on one side of the commissure, in which case there would be total blindness in one eye and temporal hemianopia in the other.

(3) Still more extensive disease is not infrequent from pressure of tumors in this region, the whole chiasma is involved, and total blindness results. The different stages in the process may often be traced in a single case from temporal hemianopia, then complete blindness in one eye with temporal hemianopia in the other, and finally complete blindness.

(4) A limited lesion of the outer part of the chiasma involves only the direct fibres passing to the temporal halves of the retinæ and inducing

blindness in the nasal field, or, as it is called, nasal hemianopia. This, of course, is extremely rare.

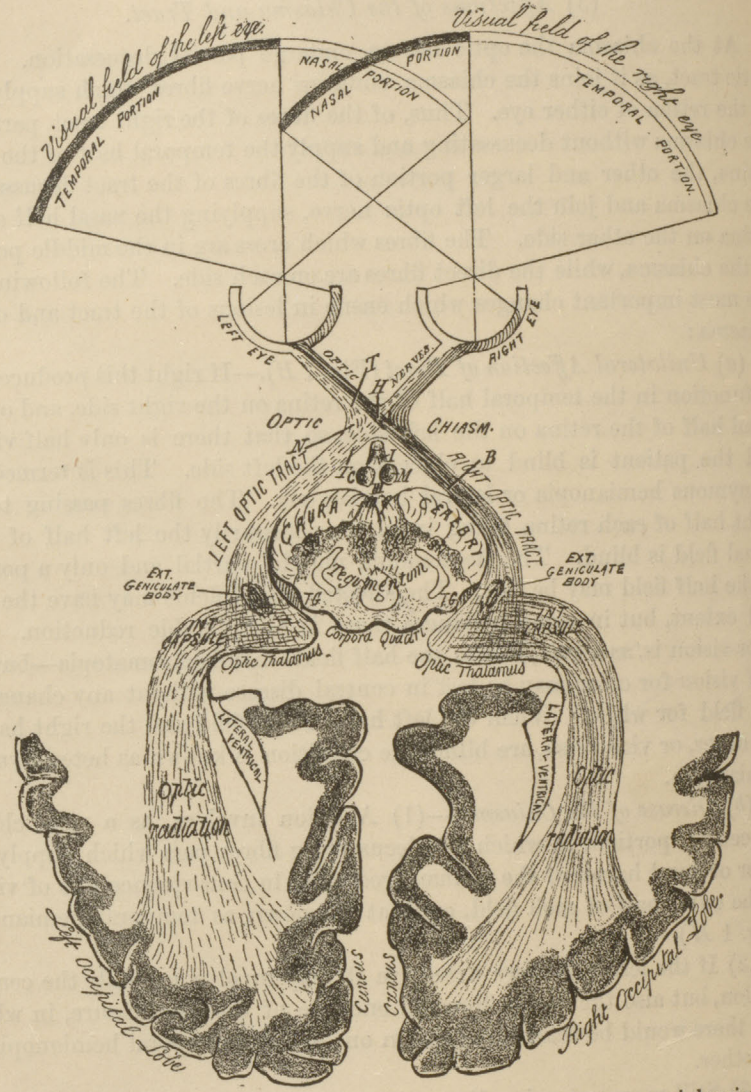


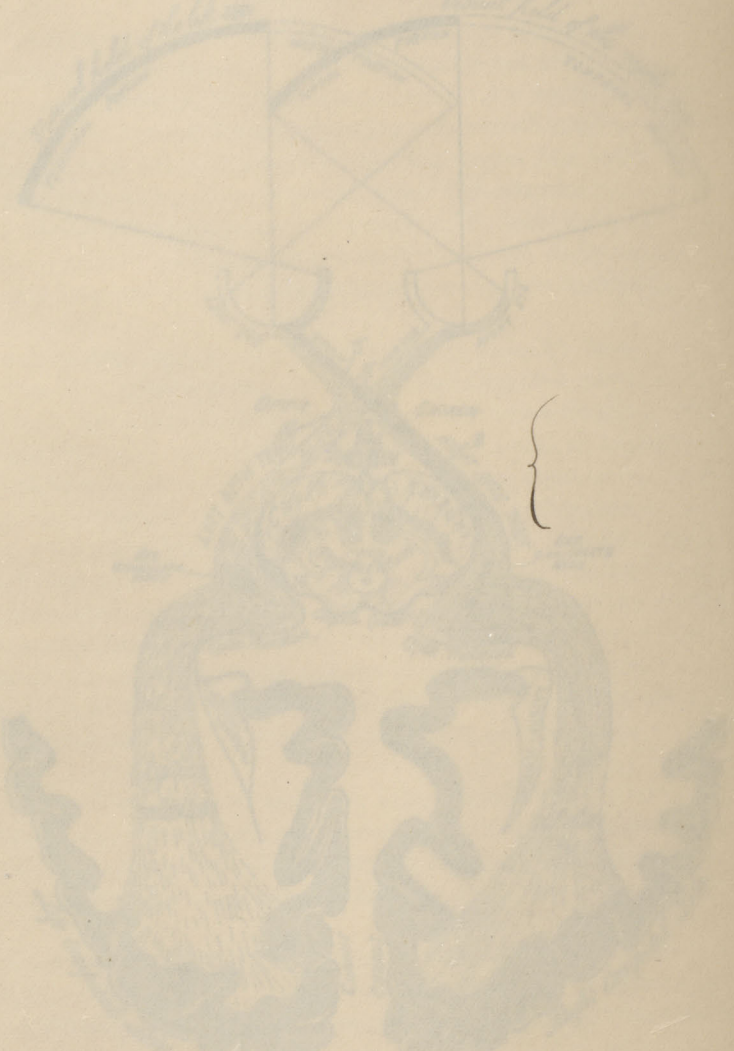
FIG. 1.—THE OPTIC AND VISUAL TRACTS (Starr). *N*, Lesion causing nasal hemianopia. *T*, Lesions causing temporal hemianopia. *H*, Lesion causing bilateral heteronymous hemianopia. *B*, Lesion of tract causing homonymous hemianopia.

(4) *Affections of the Tract and Centres.*

The optic tract crosses the crus to the hinder part of the optic thalamus and divides into two portions, one of which goes to the thalamus and external geniculate bodies and to the anterior quadrigeminal bodies. From

Double nasal hemianopia

may occur as a manifestation of tabes or in ~~rare~~ ~~multiple~~ tumors involving the inter-fibres fasciculi (Clever. 9. 3. 94.



these parts fibres pass into the posterior part of the internal capsule and enter the occipital lobe, forming the fibres of the optic radiation (Fig. 1), which terminate in and about the cuneus, the region of the visual perceptive centre. The fibres of the other division of the tract pass to the internal geniculate bodies and to the posterior quadrigeminal body. It is still held by some physiologists that the cortical visual centre is not confined to the occipital lobe alone, but embraces the occipito-angular region.

A lesion of the fibres of the optic tract anywhere between the cortical centre and the chiasma will produce lateral hemianopia. The lesion may be situated: (*a*) In the tract itself. (*b*) In the region of the thalamus and the corpora quadrigemina, into which the larger part of each tract enters. (*c*) A lesion of the fibres passing from the corpora quadrigemina to the occipital lobe. This may be either in the hinder part of the internal capsule or the white fibres of the optic radiation. (*d*) Lesion of the cuneus. Bilateral disease of the cuneus may result in total blindness. (*e*) There is clinical evidence to show that lesion of the angular gyrus may be associated with visual defect, not so often hemianopia as crossed amblyopia, dimness of vision in the opposite eye, and great contraction in the field of vision. Lesions in this region are associated with mind blindness, a condition in which there is failure to recognize the nature of objects.

The effects of lesions in the optic nerve in different situations from the retinal expansion to the brain cortex are as follows: (1) Of the optic nerve —total blindness of the corresponding eye; (2) of the optic chiasma, either temporal hemianopia, if the central part alone is involved, or nasal hemianopia, if the lateral region of each chiasma is involved; (3) lesion of the optic tract between the chiasma and the geniculate bodies, produces lateral hemianopia; (4) lesion of the central fibres of the nerve between the geniculate bodies and the cerebral cortex produces lateral hemianopia; (5) lesion of the cuneus causes lateral hemianopia; and (6) lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as mind blindness.

Diagnosis.—The student or practitioner must have a clear idea of the physiology of the nerve centres before he can appreciate the symptoms or undertake the diagnosis of lesions of the optic nerve. Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wernicke's *hemiopic pupillary inaction.* The pupil reflex depends on the integrity of the retina or receiving membrane, on the fibres of the optic nerve and tract which transmit the impulse, and the nerve centre in the geniculate bodies which receives the impression and transmits it to the third nerve along which the motor impulses pass to the iris. If a bright

light is thrown into the eye and the pupil reacts, the integrity of this reflex arc is demonstrated. It is possible in cases of lateral hemianopia so to throw the light into the eye that it falls upon the blind half of the retina. If when this is done the pupil contracts, the indication is that the reflex arc above referred to is perfect, by which we mean that the optic nerve fibres from the retinal expansion to the centre, the centre itself, and the third nerve are uninvolved. In such a case the conclusion would be justified that the cause of the hemianopia was central; that is, situated behind the geniculate bodies, either in the fibres of the optic radiation or in the visual cortical centres. If, on the other hand, when the light is carefully thrown on the hemiopic half of the retina, the pupil remains inactive, the conclusion is justifiable that there is interruption in the path between the retina and the geniculate bodies, and that the hemianopia is not central, but dependent upon a lesion situated in the tract. This test of Wernicke's is sometimes difficult to obtain. It is best performed as follows: "The patient being in a dark or nearly dark room with the lamp or gas-light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light." (Seguin.)

The significance of hemianopia varies. There is a functional hemianopia associated with migraine and hysteria. In a considerable proportion of all cases there are signs of organic brain-disease. Hemiplegia is common and the loss of power and blindness are on the same side. Thus, a lesion in the left hemisphere involving the motor tract produces right hemiplegia, and when the fibres of the optic radiation are involved in the internal capsule, there is also left lateral hemianopia, so that objects in the field of vision to the right are not perceived. Hemianæsthesia is not uncommon, owing to the close association of the sensory and visual tracts at the posterior part of the internal capsule. Certain forms of aphasia also occur in many of the cases.

III. Motor Nerves of the Eyeball.

Third Nerve.—Arising from the floor of the aqueduct of Sylvius, the nerve passes through the crus at the side of which it emerges. Passing along the wall of the cavernous sinus, it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the in-

There are two chief nuclei, the superior, on either side of the middle line in floor of the Sylvian aqueduct, just behind the nucleus of the pathetic in which are arranged from before backward and smaller independent centres for the int. rectus, the superior rectus & levator palpebræ superioris, the inferior rectus, the inf. oblique & then the pathetic. The superior nucleus also in advance of the other presents two centres one for the retaining muscle, the other for the (accommodation & pupilomotor centres) Thus the nucleus consists of the 4th. & inter. nuclear apparatus of the eye are distinct

(Astros) Review of Med 1879

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& then. Gen. features of Paral 793

usually dilated

due to affection of sympathetic nerve.

Hughlings Jackson calls

ternal and inferior recti muscles and the inferior oblique. It also supplies the ciliary muscle and the constrictor of the iris. Lesions may affect the centre or the nerve in its course and cause either paralysis or spasm.

Paralysis.—A nuclear lesion is usually associated with the disease of the centres for the other eye muscles, producing a condition of general ophthalmoplegia. More commonly the nerve itself is involved in its course, either by meningitis, gummata, or aneurism, or is attacked by neuritis, as in diphtheria and locomotor ataxia. Complete paralysis of the third nerve is accompanied by the following symptoms:

Paralysis of all the muscles, except the superior oblique and external rectus, by which the eye can be moved outward and a little downward and inward. There is divergent strabismus. There is ptosis or drooping of the upper eyelid, owing to paralysis of the levator palpebræ. The pupil is of medium size. It does not contract to light, and the power of accommodation is lost. The most striking features of this paralysis are the external strabismus, with diplopia or double vision, and the ptosis. In very many cases the affection of the third nerve is partial. Thus the levator palpebræ and the superior rectus may be involved together, or the ciliary muscles and the iris may be affected and the external muscles may escape.

There is a remarkable form of recurring oculo-motor paralysis affecting chiefly women, and involving all the branches of the nerve. In some cases the attacks have come on at intervals of a month; in others a much longer period has elapsed. The attacks may persist throughout life. They are sometimes associated with pain in the head and sometimes with migraine. Mary Sherwood has collected from the literature twenty-three cases.

Ptosis is a common and important symptom in nervous affections. We may here briefly refer to the conditions under which it may occur: (*a*) A congenital, incurable form, which is frequently seen; (*b*) the form associated with definite lesion of the third nerve, either in its course or at its nucleus. This may come on with paralysis of the superior rectus alone or with paralysis of the internal and inferior recti as well. (*c*) There are instances of complete or partial ptosis associated with cerebral lesions without any other branch of the third nerve being paralyzed. The position of the cortical centre is as yet unknown. (*d*) Hysterical ptosis, which is double and occurs with other hysterical symptoms. (*e*) Sympathetic or pseudo-ptosis is associated with symptoms of vaso-motor palsy, such as elevation of the temperature on the affected side with redness and œdema of the skin. Contraction of the pupil exists on the same side and the eyeball appears rather to have shrunk into the orbit. (*f*) In idiopathic muscular atrophy, when the face muscles are involved, there may be marked bilateral ptosis. And, lastly, in weak, deli-

cate women there is often to be seen a transient ptosis, particularly in the morning.

Among the most important of the symptoms of the third-nerve paralysis are those which relate to the ciliary muscle and iris.

Cycloplegia, paralysis of the ciliary muscle, causes loss of the power of accommodation. Distant vision is clear, but near objects cannot be properly seen. In consequence the vision is indistinct, but can be restored by the use of convex glasses. This may occur in one or in both eyes; in the latter case it is usually associated with disease in the nuclei of the nerve. Cycloplegia is an early and frequent symptom in diphtheritic paralysis and occurs also in tabes.

Iridoplegia, or paralysis of the iris, occurs in three forms (Gowers).

(*a*) *Accommodative iridoplegia*, in which the pupil does not diminish in size during the act of accommodation. To test for this the patient should look first at a distant and then at a near object in the same line of vision.

(*b*) *Reflex Iridoplegia.*—The path for the iris reflex is along the optic nerve and tract to the geniculate bodies, then to the nucleus of the third nerve, and along the trunk of this nerve to the ciliary ganglion, and so through the ciliary nerves to the eyes. Each eye should be tested separately, the other one being covered. The patient should look at a distant object in a dark part of the room; then a light is brought suddenly in front of the eye at a distance of three or four feet, so as to avoid the effect of accommodation. Loss of this iris reflex with retention of the accommodation contraction is known as the Argyll-Robertson pupil.

(*c*) *Loss of the Skin Reflex.*—If the skin of the neck is pinched or pricked the pupil dilates reflexly, the afferent impulses being conveyed along the cervical sympathetic. Erb pointed out that this skin reflex is lost usually in association with the reflex contraction, but the two are not necessarily conjoined. In iridoplegia the pupils are often small, particularly in spinal disease, as in the characteristic small pupils of tabes—spinal myosis. Iridoplegia may coexist with a pupil of medium size.

Inequality of the pupils—anisocoria—is not infrequent in progressive paresis and in tabes. It may also occur in perfectly healthy individuals.

Spasm.—Occasionally in meningitis and in hysteria there is spasm of the muscles supplied by the third nerve, particularly the internal rectus and the levator palpebræ. The clonic rhythmical spasm of the eye muscles is known as *nystagmus*, in which there is usually a bilateral, rhythmical, involuntary movement of the eyeballs. The condition is met with in many congenital and acquired brain lesions, in albinism, and sometimes in coal-miners.

Fourth Nerve.—This supplies the superior oblique muscle. In its course around the outer surface of the crus and in its passage into the

Spasm fled on 4th. suture.

At first this is only evident when the eyes are moved in the direction of the weak muscle, but may become constant by the contraction of the opposing muscle

orbit it is liable to be compressed by tumors, by aneurism or in the exudation of basilar meningitis. Its nucleus in the upper part of the fourth ventricle may be involved by tumors or undergo degeneration with the other ocular nuclei. The superior oblique muscle acts in such a way as to direct the eyeball downward and rotates it slightly. The paralysis causes defective downward and inward movement, often too slight to be noticed. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down, as in descending stairs.

Sixth Nerve.—This nerve emerges at the junction of the pons and medulla, then, passing forward, it enters the orbit and supplies the external rectus muscle. It is affected by meningitis at the base or by gummata or other tumors, and sometimes by cold. There is internal strabismus, and the eye cannot be turned outward. Diplopia occurs on looking toward the paralyzed side.

"When the nucleus is affected there is, in addition to paralysis of the external rectus, inability of the internal rectus of the opposite eye to turn that eye inwards. As a consequence of this the axes of the eyes are kept parallel and both are conjugately deviated to the opposite side, away from the side of lesion. The reason of this is that the nucleus of the sixth nerve sends fibres up in the pons to that part of the nucleus of the opposite third nerve which supplies the internal rectus. We thus have paralysis of the internal rectus without the nucleus of the third nerve being involved, owing to its receiving its nervous impulses for parallel movement from the sixth nucleus of the opposite side. As the sixth nucleus is in such proximity to the facial nerve in the substance of the pons, it is frequently found that the whole of the face on the same side is paralyzed, and gives the electrical reaction of degeneration, so that with a lesion of the *left* sixth nucleus there is conjugate deviation of both eyes to the *right*—i. e., paralysis of the left external and the right internal rectus, and sometimes complete paralysis of the *left* side of the face." (Beevor.)

General Features of Paralysis of the Motor Nerves of the Eye.—Gowers divides them into five groups:

(*a*) *Limitation of Movement.*—Thus, in paralysis of the external rectus, the eyeball is turned in by the contraction of the internal rectus and cannot be moved outward. When the paralysis is incomplete the movement is deficient in proportion to the degree of the palsy.

(*b*) *Strabismus.*—The axes of the eyes do not correspond. Thus, paralysis of the internal rectus causes a divergent squint; of the external rectus, a convergent squint. The deviation of the axis of the affected eye from parallelism with the other is called the primary deviation.

(*c*) *Secondary Deviation.*—If, while the patient is looking at an object, the sound eye is covered, so that he fixes the object looked at with the affected eye only, the sound eye is moved still further in the same di-

rection—e. g., outward—with paralysis of the opposite internal rectus. This is known as secondary deviation. It depends upon the fact that, if two muscles are acting together, when one is weak and an effort is made to contract it, the increased effort—innervation—acts powerfully upon the other muscle, causing an increased contraction.

(*d*) *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles" (Gowers). With the eyes at rest in the mid-position, an object at which we are looking is directly opposite our face. Turning the eyes to one side, we recognize that object in the middle of the field or to the side of this former position. We estimate the degree by the amount of movement of the eyes, and when the object moves and we follow it we judge of its position by the amount of movement of the eyeballs. When one ocular muscle is weak, the increased innervation gives the impression of a greater movement of the eye than has really taken place. The mind, at the same time, receives the idea that the object is further on one side than it really is, and in an attempt to touch it the finger may go beyond it. As the equilibrium of the body is in a large part maintained by a knowledge of the relation of external objects to it obtained by the action of the eye muscles, this erroneous projection resulting from paralysis disturbs the harmony of these visual impressions and may lead to giddiness —ocular vertigo.

(*e*) *Double Vision.*—This is one of the most disturbing features of paralysis of the eye muscles. The visual axes do not correspond, so that there is a double image—diplopia. That seen by the sound eye is termed the true image; that by the paralyzed eye, the false. In simple or homonymous diplopia the false image is "on the same side of the other as the eye by which it is seen." In crossed diplopia it is on the other side. In convergent squint the diplopia is simple; in divergent it is crossed.

Ophthalmoplegia.—Under this term is described a chronic progressive paralysis of the ocular muscles. Two forms are recognized—ophthalmoplegia *externa* and ophthalmoplegia *interna*. The conditions may occur separately or together and are described by Gowers under nuclear ocular palsy.

Ophthalmoplegia Externa.—The condition is one of more or less complete palsy of the external muscles of the eyeball, due usually to a slow degeneration in the nuclei of the nerves, but sometimes to pressure of tumors or to basilar meningitis. It is often but not necessarily associated with ophthalmoplegia interna. Siemerling, in the recent monograph in which he has analyzed the material (eight cases) left by the late Prof. Westphal, states that sixty-two cases are on record. In only eleven of these could syphilis be positively determined. The levator muscles of the eyelids and the superior recti are first involved, and gradually the other

Elaborate.

muscles, so that the eyeballs are fixed and the eyelids droop. There is sometimes slight protrusion of the eyeballs. The disease is essentially chronic and may last for many years. It is found particularly in association with general paralysis, locomotor ataxia, and in progressive muscular atrophy. Mental disorders were present in eleven of the sixty-two cases. With it may be associated atrophy of the optic nerve and affections of other cranial nerves. Occasionally, as noted by Bristowe, it may be functional.

Ophthalmoplegia Interna.—Jonathan Hutchinson applied this term to a progressive paralysis of the internal ocular muscles, causing loss of pupillary action and the power of accommodation. When the internal and external muscles are involved the affection is known as total ophthalmoplegia, and in a majority of the cases the two conditions are associated. In some instances the internal form may depend upon disease of the ciliary ganglion.

While, as a rule, ophthalmoplegia is a chronic process, there is an acute form associated with hæmorrhagic softening of the nuclei of the ocular muscles. There is usually marked cerebral disturbance. It was to this form that Wernicke gave the name polio-encephalitis superior.

Treatment of Ocular Palsies.—It is important to ascertain, if possible, the cause. The forms associated with locomotor ataxia are obstinate, and resist treatment. Occasionally, however, a palsy, complete or partial, may pass away spontaneously. The group of cases associated with chronic degenerative changes, as in progressive paresis and bulbar paralysis, is little affected by treatment. On the other hand, in syphilitic cases, mercury and iodide of potassium are indicated and are often beneficial. Arsenic and strychnia, the latter hypodermically, may be employed. In any case in which the onset is acute, with pain, hot fomentations and counter-irritation or leeches applied to the temple give relief. The direct treatment by electricity has been extensively employed, but probably without any special effect. The diplopia may be relieved by the use of prisms, or it may be necessary to cover the affected eye with an opaque glass.

IV. Fifth Nerve.

Paralysis may result from: (*a*) Disease of the pons, particularly hæmorrhage or patches of sclerosis. (*b*) Injury or disease at the base of the brain. Fracture rarely involves the nerve; on the other hand, meningitis, acute or chronic, and caries of the bone are not uncommon causes. (*c*) The branches may be affected as they pass out—the first division by tumors pressing on the cavernous sinus or by aneurism; the second and third divisions by growths which invade the spheno-maxillary fossa. (*d*) Primary neuritis, which is rare.

Symptoms.—(*a*) *Sensory Portion.*—Paralysis of the fifth nerve causes loss of sensation in the parts supplied, including the half of the

face, the corresponding side of the head, the conjunctiva, the mucosa of the lips, tongue, hard and soft palate, and of the nose of the same side. The anæsthesia may be preceded by tingling or pain. The muscles of the face are also insensible and the movements may be slower. The sense of smell is interfered with. There is ~~loss~~ of the sense of taste. There are, in addition, *trophic* changes; the salivary, lachrymal, and buccal secretions may be lessened, abrasions of the mucous membranes heal slowly, and the teeth may become loose. The eye inflames, the corneæ become cloudy and may ulcerate. ~~These latter symptoms occur only~~ when the Gasserian ganglion is affected, ~~as the nerve itself may be involved for years without~~ producing ophthalmia. Herpes may develop in the region supplied by the nerve and is usually associated with much pain. It is most common in the upper branch of the nerve. The pain which follows the herpes may be peculiarly enduring, lasting for months or years (Gowers).

(*b*) *Motor Portion.*—The inability to use the muscles of mastication on the affected side is the distinguishing feature of paralysis of this portion of the nerve. It is recognized by placing the finger on the masseter and temporal muscles, and, when the patient closes the jaw, the feebleness of their contraction is noted. If paralyzed, the external pterygoid cannot move the jaw toward the unaffected side; and when depressed, the jaw deviates to the paralyzed side. The motor paralysis of the fifth nerve is almost invariably a result of involvement of the nerve after it has left the nucleus. Cases, however, have been associated with cortical lesions. Hirt concludes, from his case, that the motor centre for the trigeminus is in the neighborhood of the lower third of the ascending frontal convolution.

Spasm of the Muscles of Mastication.—Trismus, the masticatory spasm of Romberg, may be tonic or clonic, and is either an associated phenomenon in general convulsions or, more rarely, an independent affection. In the tonic form the jaws are kept close together—lock-jaw—or can be separated only for a short space. The muscles of mastication can be seen in contraction and felt to be hard and the spasm is often painful. This tonic contraction is an early symptom in tetanus, and is sometimes seen in tetany. A form of this tonic spasm occurs in hysteria. Occasionally trismus follows exposure to cold, and is said to be due to reflex irritation from the teeth, the mouth, or caries of the jaw. It may also be a symptom of organic disease due to irritation near the motor nucleus of the fifth nerve.

Clonic spasm of the muscles supplied by the fifth occurs in the form of rapidly repeated contractions, as in "chattering teeth." This is rare apart from general conditions, though cases are on record, usually in women late in life, in whom this isolated clonic spasm of the muscles of the jaw has been found. In another form of clonic spasm sometimes seen in chorea, there are forcible single contractions. Gowers mentions an instance of its occurrence as an isolated affection.

(*c*) *Gustatory.*—Loss of the sense of taste in the anterior two thirds of

disturbance

It was formerly held that these symptoms only occurred
when
but of late years this has been completely
removed for Schmidtale without producing
any trophic disturbance.

to the chorda tympani; runs
with the facial to the geniculate ganglion and
pass by the greater petrosal nerve to the
2nd branch of the 5th, but take investing
Look at
see under that they may reach the brain thro
the intermaxillary nerve of Meckel.

Operative treatment.

(Von Graefe)

In rare instances these muscles are
paralyzed (Beevor) cure at N H P & S.

the tongue, as a rule, follows paralysis of the fifth nerve. The gustatory fibres pass from the chorda tympani to the lingual branch of the fifth. Disease of the fifth nerve is, however, not always associated with loss of taste in the anterior part of the tongue, in which case either the taste fibres escape, or the disease is within the pons where these fibres are separate from those of sensation.

The *diagnosis* of disease of the trifacial nerve is rarely difficult. It must be remembered that the preliminary pain and hyperæsthesia are sometimes mistaken for neuralgia. The loss of sensation and the palsy of the muscles of mastication are readily determined.

Treatment.—When the pain is severe morphia may be required and local applications are useful. If there is a suspicion of syphilis, appropriate treatment should be given. Faradization is sometimes beneficial.

V. Facial Nerve.

Paralysis (*Bell's Palsy*).—The *portio dura* of the seventh pair may be paralyzed by (1) lesions of the cortex—supranuclear palsy; (2) lesions of the nucleus itself; or (3) involvement of the nerve trunk in its tortuous course within the pons and through the wall of the skull.

I. *Supranuclear Paralysis*, due to lesion of the cortex or of the facial fibres in the corona radiata or internal capsule, is, as a rule, associated with hemiplegia. It may be caused by tumors, abscess, chronic inflammation, or softening in the region of the internal capsule. It is distinguished from the peripheral form by two well-marked characters—the persistence of the normal electrical excitability of both nerves and muscles and the absence of involvement of the upper branches of the nerve, so that the orbicularis palpebrarum and frontalis muscle are spared. A third difference is that in this form the voluntary movements are more impaired than the emotional. There are instances of cortical facial paralysis—monoplegia facialis—associated with lesions in the centre for the face muscles in the lower Rolandic region. Isolated paralysis, due to involvement of the nerve fibres in their path to the nucleus, is uncommon. In the great majority of cases supranuclear facial paralysis is part of a hemiplegia. Paralysis is on the same side as that of the arm and leg because the facial muscles bear precisely the same relation to the cortex as the spinal muscles. The nuclei of origin on either side of the middle line in the medulla are united by decussating fibres with the cortical centre on the opposite side (see Fig. 3).

II. The *nuclear paralysis* caused by lesions of the nerve centre in the medulla is not common alone; but is seen occasionally in tumors, chronic softening, and hæmorrhage. In rare instances of anterior polio-myelitis the facial nucleus is affected. In diphtheria this centre may also be involved. The symptoms are practically similar to those of an affection of the nerve fibre itself—infranuclear paralysis.

III. *Involvement of the Nerve Trunk.*—Paralysis may result from:

(*a*) Involvement of the nerve as it passes through the pons—that is, between its nucleus in the floor of the fourth ventricle and the point of emergence in the postero-lateral aspect of the pons. The specially interesting feature in connection with involvement of this part is the production of what is called alternating or *cross paralysis*, the face being involved on the same side as the lesion, and the arm and leg on the opposite side, since the motor path is involved above the point of decussation in the medulla (Fig. 3, *z*). This occurs only when the lesion is in the lower section of the pons. A lesion in the upper division involves the fibres not of the outgoing nerve on the same side, but of the ~~nerve of the other side, which has crossed and is ascending to the hemisphere~~. In this case there would of course be, as in hemiplegia, paralysis of the face and limbs on the side opposite to the lesion. The palsy, too, would resemble the cerebral form, involving only the lower fibres of the facial nerve.

(*b*) The nerve may be involved at its point of emergence by tumors, gummata, meningitis, or occasionally may be injured in fracture of the base.

(*c*) In passing through the Fallopian canal the nerve may be involved in disease of the ear, particularly by caries of the bone in otitis media. This is a common cause in children.

(*d*) As the nerve emerges from the styloid foramen it is exposed to injuries and blows which not infrequently cause paralysis. The fibres may be cut in the removal of tumors in this region, or the paralysis may be caused by pressure of the forceps in an instrumental delivery.

(*e*) Exposure to cold is the most common cause of facial paralysis, inducing a neuritis of the nerve within the Fallopian canal. It is sometimes termed rheumatic neuritis, but there is no evidence that it is specially associated with the rheumatic poison.

Facial diplegia is a rare condition occasionally found in affections at the base of the brain, lesions in the pons, simultaneous involvement of the nerves in ear disease, and in diphtheritic paralysis. Disease of the nuclei or symmetrical involvement of the cortex might also produce it.

Symptoms.—In the peripheral facial paralysis all the branches of the nerve are involved. The face on the affected side is immobile and can neither be moved at will nor participate in any emotional movements. The skin is smooth and the wrinkles are effaced, a point particularly noticeable on the forehead of elderly persons. The eye cannot be closed, the lower lid droops, and the eye waters. On the affected side the angle of the mouth is lowered, and in drinking the lips are not kept in close apposition to the glass, so that the liquid is apt to run out. In smiling or laughing the contrast is most striking, as the affected side does not move, which gives a curious unequal appearance to the two sides of the face. The eye cannot be closed and the forehead cannot be wrinkled. On asking a patient to show his upper teeth, the angle of the mouth is not raised. In

fibres from the hemispheres before they have crossed to the nucleus of the opposite side

(f) Syphilis is an important frequent cause, and the paralysis may develop early with the secondary symptoms.

The face on the affected side may be swollen.

all these movements the face is drawn to the sound side by the action of the muscles. Speaking may be slightly interfered with, owing to the imperfection in the formation of the labial sounds. Whistling cannot be performed. In chewing the food, owing to the paralysis of the buccinator, particles collect on the affected side. The paralysis of the nasal muscles is seen on asking the patient to sniff. Owing to the fact that the lips are drawn to the sound side, the tongue, when protruded, looks as if it were pushed to the paralyzed side; but on taking its position from the incisor teeth, it will be found to be in the middle line. The reflex movements are lost in this peripheral form. It is usually stated that the palate is paralyzed on the same side and that the uvula deviates. Both Gowers and Hughlings Jackson deny the existence of this involvement in the great majority of cases, and Horsley and Beevor have shown that these parts are innervated by the accessory nerve to the vagus.

When the nerve is involved within the canal between the genu and the origin of the chorda tympani, the sense of taste may be lost in the anterior part of the tongue on the affected side. When the nerve is damaged outside the skull the sense of taste is unaffected. Hearing is often impaired in facial paralysis, most commonly by preceding ear disease. The paralysis of the stapedius muscle may lead to increased sensitiveness to musical notes. Herpes is sometimes associated with facial paralysis. Pain is not common, but there may be neuralgia about the ear.

The *electrical reactions*, which are those of a peripheral palsy, have considerable importance from a prognostic standpoint. Erb's rules are as follows: If there is no change, either faradic or galvanic, the prognosis is good and recovery takes place in from fourteen to twenty days. If the faradic and galvanic excitability of the nerve is only lessened and that of the muscle increased to the galvanic current and the contraction formula altered (the contraction sluggish AnC>CC), the outlook is relatively good and recovery will probably take place in from four to six weeks; occasionally in from eight to ten. When the reaction of degeneration is present—that is, if the faradic and galvanic excitability of the nerves and the faradic excitability of the muscles are lost and the galvanic excitability of the muscle is quantitatively increased and qualitatively changed, and if the mechanical excitability is altered—the prognosis is relatively unfavorable and the recovery may not occur for two, six, eight, or even fifteen months.

The *course* of facial paralysis is usually favorable. The onset in the form following cold is very rapid, developing perhaps within twenty-four hours, but rarely is the paralysis permanent. On the other hand, in the paralysis from injury, as by a blow on the mastoid process, the paralysis may remain. When permanent the muscles are entirely toneless. In some instances contracture develops as the voluntary power returns, and the natural folds and the wrinkles on the affected side may be deepened, so that on

looking at the face one at first may have the impression that the affected side is the sound one. This is corrected at once on asking the patient to smile, when it is seen which side of the face has the most active movement.

The *diagnosis* of facial paralysis is usually easy. The distinction between peripheral and central is based on facts already mentioned.

Treatment.—In the cases which result from cold and are probably due to neuritis within the bony canal, hot applications first should be made; subsequently the thermo-cautery may be used lightly at intervals of a day or two over the mastoid process, or small blisters applied. If the ear is diseased, free discharge for the secretion should be obtained. The continuous current may be employed to keep up the nutrition of the muscles. The positive pole should be placed behind the ear, the negative one along the zygomatic and other muscles. The application can be made daily for a quarter of an hour and the patient can readily be taught to make it himself before the looking-glass. Massage of the muscles of the face is also useful.

A course of iodide of potassium may be given even when there is no indication of syphilis.

Spasm.—The spasm may be limited to a few or involve all the muscles innervated by the facial nerve and may be unilateral or bilateral.

It is known also by the name of mimic spasm or of convulsive tic. Several different affections are usually considered under the name of facial or mimic spasm, but we shall here speak only of the simple spasm of the facial muscles, either primary or following paralysis, and shall not include the cases of habit spasm in children, or the *tic convulsif* of the French.

Gowers recognizes two classes—one in which there is an organic lesion, and an idiopathic form. It is thought to be due also to reflex causes, such as the irritation from carious teeth or the presence of intestinal worms. The disease usually occurs in adults, whereas the habit spasm and the *tic convulsif* of the French, often confounded with it, are most common in children. True mimic spasm occasionally comes on in childhood and persists. In the case of a school-mate, the affection was marked as early as the eleventh or twelfth year and still continues. When the result of organic disease there has usually been a lesion of the centre in the cortex, as in the case reported by Berkeley, or pressure on the nerve at the base of the brain by aneurism or tumor.

Symptoms.—The spasm may involve only the muscles around the eye—blepharospasm—in which case there is constant, rapid, quick action of the orbicularis palpebrarum, which, in association with photophobia, may be tonic in character. More commonly the spasm affects the lateral facial muscles with those of the eye and there is constant twitching of the side of the face with partial closure of the eye. The frontalis is rarely in-

volved. In aggravated cases the depressors of the angle of the mouth, the levator menti, and the platysma myoides are affected. This spasm is confined to one side of the face in a majority of cases, though it may extend and become bilateral. It is increased by emotional causes and involuntary movements of the face. As a rule, it is painless, but there may be tender points on the course of the fifth nerve, particularly the supraorbital branch. Tonic spasm of the facial muscle may follow paralysis, and is said to result occasionally from cold.

The outlook in facial spasm is always dubious. A majority of the cases persist for years and are incurable.

Treatment.—Sources of irritation should be looked for and removed. When a painful spot is present over the fifth nerve, blistering or the application of the thermo-cautery may relieve it. Hypodermic injections of strychnia may be tried, but are of doubtful benefit. Weir Mitchell recommends the freezing of the cheek for a few minutes daily or every second day with the spray, and this, in some instances, is beneficial. Often the relief is transient; the cases return, and at every clinic may be seen half a dozen or more of such patients who have run the gamut of all measures without material improvement. Operative interference may be resorted to in severe cases, although not much can be expected of it.

VI. Auditory Nerve.

This nerve, forming the *portio mollis* of the seventh pair, enters the internal auditory meatus, and divides into the cochlear and vestibular branches. The cortical centre for hearing is in the temporo-sphenoidal lobe. Primary disease of the auditory nerve in its centre or intracranial course is uncommon. More frequently the terminal branches are affected within the labyrinth.

(*a*) *Affection of the Cortical Centre.*—In the monkey, experiments indicate that the first temporal gyri represent the centre for hearing. In man the cases of disease indicate that it has the same situation, as destruction of this gyrus on the left side results in word-deafness, which may be defined as an inability to understand the meaning of words, though they may still be heard as sounds. The central fibres of the auditory nerve between the cortical centre and the nucleus in the fourth ventricle may be involved and produce deafness. This has resulted from the presence of a tumor in the corpora quadrigemina, and may be associated with a lesion of the internal capsule.

(*b*) *Lesions of the nerve at the base* of the brain may result from the pressure of tumors, meningitis (particularly the cerebro-spinal form), hæmorrhage, or traumatism. A primary degeneration of the nerve may occur in locomotor ataxia. Nuclear disease is rare. By far the most interesting form results from epidemic cerebro-spinal meningitis, in which the

nerve is frequently involved, causing permanent deafness. In young children the condition results in deaf-mutism.

(*c*) In a majority of the cases associated with auditory-nerve symptoms the lesion is in the *labyrinth*, either primary or the result of extension of disease of the middle ear. Three groups of symptoms may be produced—hyperæsthesia and irritation, diminished function or nervous deafness, and vertigo.

(1) *Hyperæsthesia and Irritation.*—This may be due to altered function of the centre as well as of the nerve ending. True hyperæsthesia—hyperacusis—is a condition in which sounds, sometimes even those inaudible to other persons, are heard with great intensity. It occurs in hysteria and occasionally in cerebral disease. As already mentioned, in paralysis of the stapedius low notes may be heard with intensity. In dysæsthesia, or dysacusis, ordinary sounds cause an unpleasant sensation, as commonly happens in connection with headache, when ordinary noises are badly borne.

Tinnitus aurium is a term employed to designate certain subjective sensations of ringing, roaring, ticking, and whirring noises in the ear. It is a very common and often a distressing symptom. It is associated with many forms of ear disease and may result from pressure of wax on the drum. It is rare in organic disease of the central connections of the nerve. Sudden intense stimulation of the nerve may cause it. A form not uncommonly met with in medical practice is that in which the patient hears a continual *bruit* in the ear, and the noise has a systolic intensification, usually on one side. I have twice been consulted by physicians for this condition under the belief that they had an internal aneurism. It occurs in conditions of anæmia and neurasthenia. Subjective noises in the ear may precede an epileptic seizure and are sometimes present in migraine. In whatever form tinnitus exists, though slight and often regarded as trivial, it occasions great annoyance and often mental distress, and has even driven patients to suicide.

The *diagnosis* is readily made; but it is often extremely difficult to determine upon what condition the tinnitus depends. The relief of constitutional states, such as anæmia, neurasthenia, or gout, may result in cure. A careful local examination of the ear should always be made. One of the most worrying forms is the constant clicking, sometimes audible many feet away from the patient, and due probably to clonic spasm of the muscles connected with the Eustachian tube or of the levator palati. The condition may persist for years unchanged, and then disappear suddenly. The pulsating forms of tinnitus, in which the sound is like that of a systolic *bruit*, are almost invariably subjective, and nothing is audible with the stethoscope. It is to be remembered that in children there is a systolic brain murmur, best heard over the ear, and in some instances is heard in the adult.

(2) *Diminished Function or Nervous Deafness.*—In testing for nervous

deafness, if the tuning-fork cannot be heard when placed near the meatus, but the vibrations are audible by placing the foot of the tuning-fork against the temporal bone, the conclusion may be drawn that the deafness is not due to involvement of the nerve. The vibrations are conveyed through the temporal bone to the cochlea and vestibule. The watch may be used for the same purpose, and if the meatus is closed and the watch is heard better in contact with the mastoid process than when opposite the open meatus, the deafness is probably not nervous. Practically, disturbance of the function of the auditory nerve is not a very frequent symptom in brain-disease, but in all cases the function of the nerve should be carefully tested

(3) **Auditory Vertigo—Menière's Disease.**—In 1861 Menière, a French physician, described an affection characterized by noises in the ear, vertigo (which might be associated with loss of consciousness), vomiting, and, in many cases, progressive loss of hearing. The term is now used to include all cases of sudden vertigo accompanied by noises in the ear and deafness. The frequency of vertigo with ear symptoms is striking. Thus, of 106 cases noted by Gowers, in which there was definite vertigo, in 94 ear symptoms were present, either tinnitus or deafness or both.

Symptoms.—The attack usually sets in suddenly with a buzzing noise in the ears and the patient feels as if he was reeling or staggering. He may feel himself to be reeling, or the objects about him may seem to be turning, or the phenomena may be combined. The attack is often so abrupt that the patient falls, though, as a rule, he has time to steady himself by grasping some neighboring object. There may be slight but transient loss of consciousness. In a few minutes, or even less, the vertigo passes off and the patient becomes pale and nauseated, a clammy sweat breaks out on the face, and vomiting may follow.

The deafness, which is always of a nervous character, may be in only one ear and is never complete. The tinnitus is described as either a roaring or a throbbing sound. Ocular symptoms may be present; thus, jerking of the eyeballs or nystagmus may develop during the attack, or diplopia.

Labyrinthine vertigo is paroxysmal, coming on at irregular intervals. Sometimes weeks or months may elapse between the attacks; in other cases there may be several attacks in a day. As a rule, the patients have no affection of the middle ear. The disease rarely occurs in young persons, is most frequent after the fortieth year, and is more common in men than in women.

The pathology of the disease has been much discussed. There are two theories concerning its origin—one, that it is due to affection of the labyrinth itself, which causes a disturbance of equilibrium, such as is proved by experiment to be associated with lesion of the semicircular canals; the other that it is really a trouble involving the centres presiding over hearing and equilibration.

It has also been held to be a vaso-motor neurosis of the vessels of the labyrinth. The condition of the labyrinth in these cases is variable. Acute disease with hæmorrhage has been described, or slow progressive degeneration of the nerves. Giddiness and vomiting may, however, be produced by irritation in other parts of the ear; thus, there are instances in which pressure on the drum or irritation of the external meatus is followed by an attack of giddiness and vomiting.

Diagnosis.—The combination of tinnitus with giddiness, with or without gastric disturbance, is sufficient to establish a diagnosis. There are other forms of vertigo from which it must be distinguished. The form known as gastric vertigo, which is associated with dyspepsia and occurs most commonly in persons of middle age, is, as a rule, readily distinguished by the absence of tinnitus or evidences of disturbance in the function of the auditory nerve. This variety of vertigo is much less common than Trousseau's description would lead us to believe.

The cardio-vascular vertigo, one of the most common forms, occurs in cases of valvular disease, particularly aortic insufficiency, and as frequently in arterio-sclerosis.

There is a remarkable form of vertigo described by Gerlier, which is characterized by attacks of paretic weakness of the extremities, falling of the eyelids, remarkable depression, but with retention of consciousness. It attacks only men, and has occurred in epidemic form among laborers in the canton of Geneva.

Aural vertigo must be carefully distinguished from attacks of *petit mal*, or, indeed, of definite epilepsy. It is rare in *petit mal* to have noises in the ear or actual giddiness, but in the aura preceding an epileptic attack the patient may feel giddy. Giddiness and transient loss of consciousness may be associated with organic disease of the brain, more particularly with tumor. Vomiting also may be present. A careful investigation of the symptoms will usually lead to a correct diagnosis.

The outlook in Menière's disease is uncertain. While many cases recover completely, in others deafness results and the attacks recur at shorter intervals. In aggravated cases the patient constantly suffers from vertigo and may even be confined to his bed.

Treatment.—Bromide of potassium, in twenty-grain doses three times a day, is sometimes beneficial. If there is a history of syphilis, the iodide should be administered. The salicylates are recommended, and Charcot advises quinine to cinchonism. In cases in which there is increase in the arterial tension nitroglycerine may be given, at first in very small doses, but increasing gradually. It is not specially valuable in Menière's disease, but in the cases of giddiness in middle-aged men and women associated with arterio-sclerosis it sometimes acts very satisfactorily.

VII. Glosso-pharyngeal Nerve.

This nerve contains both motor and sensory fibres and is also a nerve of the special sense of taste to the tongue. It supplies, by its motor branches, the stylo-pharyngeus and the middle constrictor of the pharynx. The sensory fibres are distributed to the upper part of the pharynx.

Symptoms.—Of nuclear disturbance we know very little. The pharyngeal symptoms of bulbar paralysis are probably associated with involvement of the nuclei of this nerve. Lesion of the nerve trunk itself is rare, but it may be compressed by tumors or involved in meningitis. Disturbance of the sense of taste may result from loss of function of this nerve, in which case it is chiefly in the posterior part of the tongue and soft palate. Gowers, however, states that there is no case on record in which loss of taste in these regions has been produced by disease of the roots of the glosso-pharyngeal; whereas, on the other hand, disease of the root of the fifth nerve may cause loss of taste on the back as well as the front of the tongue, as if the taste fibres of the glosso-pharyngeal came from the fifth.

The general disturbances of the sense of taste may here be briefly referred to. Loss of the sense of taste—*ageusia*—may be caused by disturbance of the peripheral end organs, as in affections of the mucosa of the tongue. This is very common in the dry tongue of fever or the furred tongue of dyspepsia, under which circumstances, as the saying is, everything tastes alike. Strong irritants too, such as pepper, tobacco, or vinegar, may dull or diminish the sense of taste. Complete loss may be due to involvement of the nerves either in their course or in the centres. Disturbance in the sense of taste is most commonly seen in involvement of the fifth nerve, and it may be that this nerve alone subserves the function. Perversion of the sense of taste—*parageusis*—is rarely found, except as an hysterical manifestation and in the insane. Increased sensitiveness is still more rare. There are occasional subjective sensations of taste, occurring as an aura in epilepsy or as part of the hallucinations in the insane.

To test the sense of taste the patient's eyes should be closed and small quantities of various substances applied. The sensation should be perceived before the tongue is withdrawn. The following are the most suitable tests: For bitter, quinine; for sweetness, a strong solution of sugar or saccharin; for acidity, vinegar; and for the saline test, common salt. One of the most important tests is the feeble galvanic current, which gives the well-known metallic taste.

VIII. Pneumogastric Nerve.

The vagus nerve has an important and extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach. The nerve may be involved at its nucleus with the spinal accessory and the hypoglossal, forming what is known as bulbar paralysis. It may be com-

pressed by tumors or aneurism, or in the exudation of meningitis, simple or syphilitic. In its course in the neck the trunk may be involved by tumors or in wounds. It has been tied in ligature of the carotid, and has been cut in the removal of deep-seated tumors. The trunk may be attacked by neuritis.

The affections of the vagus are best considered in connection with the distribution of the separate nerves.

(*a*) **Pharyngeal Branches.**—In combination with the glosso-pharyngeal the branches from the vagus form the pharyngeal plexus, from which the muscles and mucosa of the pharynx are supplied. In *paralysis* due to involvement of this either in the nuclei, as in bulbar paralysis, or in the course of the nerve, as in diphtheritic neuritis, there is difficulty in swallowing and the food is not passed on into the œsophagus. If the nerve on one side only is involved, the deglutition is not much impaired. In these cases the particles of food frequently pass into the larynx, and, when the soft palate is involved, into the posterior nares.

Spasm of the pharynx is always a functional disorder, usually occurring in hysterical and nervous people. Gowers mentions a case of a gentleman who could not eat unless alone, on account of the inability to swallow in the presence of others from spasm of the pharynx. This spasm is a well-marked feature in hydrophobia, and I have seen it in a case of pseudo-hydrophobia.

(*b*) **Laryngeal Branches.**—The superior laryngeal nerve supplies the mucous membrane of the larynx above the cords and the crico-thyroid muscle. The inferior or recurrent laryngeal curves around the arch of the aorta on the left side and the subclavian artery on the right, passes along the trachea and supplies the mucosa below the cords and all the muscles of the larynx except the crico-thyroid and the epiglottidean. Experiments have shown that these motor nerves of the pneumogastric are all derived from the spinal accessory. The remarkable course of the recurrent laryngeal nerves renders them liable to pressure by tumors within the thorax, particularly by aneurism. The following are the most important forms of paralysis:

(1) *Bilateral Paralysis of the Abductors.*—In this condition, the posterior crico-arytenoids are involved and the glottis is not opened during inspiration. The cords may be close together in the position of phonation, and during inspiration may be brought even nearer together by the pressure of air, so that there is only a narrow chink through which the air whistles with a noisy stridor. This dangerous form of laryngeal paralysis occurs occasionally as a result of cold, or may follow a laryngeal catarrh. The posterior muscles have been found degenerated when the others were healthy. The condition may be produced by pressure upon both vagi, or upon both recurrent nerves. As a central affection it occurs in tabes and bulbar paralysis, but may occur also in hysteria. The characteristic symptoms are inspiratory stridor with unimpaired phonation. Possibly,

as Gowers suggests, many cases of so-called hysterical spasm of the glottis are in reality abductor paralysis.

(2) *Unilateral Abductor Paralysis.*—This frequently results from the pressure of tumors or involvement of one recurrent nerve. Aneurism is by far the most common cause, though on the right side the nerve may be involved in thickening of the pleura. The symptoms are hoarseness or roughness of the voice, such as is so common in aneurism. Dyspnœa is not often present. The cord on the affected side does not move in inspiration. Subsequently the adductors may also become involved, in which case the phonation is still more impaired.

(3) *Adductor Paralysis.*—This results from involvement of the lateral crico-arytenoid and the arytenoid muscle itself. It is common in hysteria, particularly of women, and causes the hysterical aphonia, which may come on suddenly. It may result from catarrh of the larynx or from overuse of the voice. In laryngoscopic examination it is seen, on attempt at phonation, that there is no power to bring the cords together. In this connection the following table from Gowers work will be found valuable to the student:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the Muscles of the Larynx.—In this the adductor muscles are involved. It is not an uncommon affection in children, and has already been referred to as laryngismus stridulus. Paroxysmal attacks of laryngeal spasm are rare in the adult, but cases are described in which the patient, usually a young girl, wakes at night in an attack of intense dyspnœa, which may persist long enough to produce cyanosis. Liveing states that they may replace attacks of migraine. They occur in a characteristic form in locomotor ataxia, forming the so-called laryngeal crises. There is a condition known as spastic aphonia, in which, when the patient attempts to speak, phonation is completely prevented by a spasm.

Disturbance of the sensory nerves of the larynx is rare.

Anæsthesia may occur in bulbar paralysis and in diphtheritic neuritis—a serious condition, as portions of food may enter the windpipe. It is usually associated with dysphagia and is sometimes present in hysteria. Hyperæsthesia of the larynx is rare.

(*c*) **Cardiac Branches.**—The cardiac plexus is formed by the union of branches of the vagi and of the sympathetic nerves. The vagus fibres subserve motor, sensory, and probably trophic functions.

(1) *Motor.*—The fibres which inhibit, control, and regulate the cardiac action pass in the vagi. Irritation may produce slowing of the action. Czermak could slow or even arrest the heart's action for a few beats by pressing a small tumor in his neck against one pneumogastric nerve, and it is said that the same can be produced by forcible bilateral pressure on the carotid canal. There are instances in which persons appear to have had voluntary control over the action of the heart. The most remarkable instance was that of Colonel Townsend, who could slow the action of the heart at will. Retardation of the heart's action has also followed accidental ligature of one vagus. Irritation at the nuclei may also be accompanied by extreme slowness. The condition of brachycardia may be associated with a neurosis of this nerve. On the other hand, when there is complete paralysis of the vagi, the inhibitory action may be abolished and the acceleratory influences have full sway. The heart's action is then greatly increased. This is seen in some instances of diptheritic neuritis and in involvement of the nerve by tumors, or its accidental removal or ligature. Complete loss of function of one vagus may, however, not be followed by any symptoms.

(2) *Sensory* symptoms on the part of the cardiac branches are very varied. Normally, the heart's action proceeds regularly without the participation of consciousness, but the unpleasant feelings and sensations of palpitation and pain are conveyed to the brain through this nerve. How far the fibres of the pneumogastric are involved in angina it is impossible to say. The various disturbances of sensation are described under the cardiac neuroses.

(*d*) **Pulmonary Branches.**—We know very little of the pulmonary branches of the vagi. The motor fibres are stated to control the action of

the bronchial muscles, and it has long been held that asthma may be a neurosis of these fibres. The various alterations in the respiratory rhythm are probably due more to changes in the centre than in the nerves themselves.

(*e*) **Gastric and Œsophageal Branches.**—The muscular movements of these parts are presided over by the vagi and vomiting is induced through them, usually reflexly, but also by direct irritation, as in meningitis. Spasm of the œsophagus generally occurs with other nervous phenomena. Gastralgia may sometimes be due to cramp of the stomach, but is more commonly a sensory disturbance of this nerve, due to direct irritation of the peripheral ends, or is a neuralgia of the terminal fibres. Hunger is said to be a sensation aroused by the pneumogastric, and some forms of nervous dyspepsia probably depend upon disturbed function of this nerve. The severe gastric crises which occur in locomotor ataxia are due to central irritation of the nuclei. Some describe exophthalmic goitre under lesions of the vagi.

IX. Spinal Accessory Nerve.

Paralysis.—The smaller or internal part of this nerve joins the vagus and is distributed through it to the laryngeal muscles. The larger external part is distributed to the sterno-mastoid and trapezius muscles.

The nuclei of the nerve, particularly of the accessory part, may be involved in bulbar paralysis. The nuclei of the external portion, situated as they are in the cervical cord, may be attacked in progressive degeneration of the motor nuclei of the cord. The nerve may be involved in the exudation of meningitis, or be compressed by tumors, or in caries. The *symptoms* of paralysis of the accessory portion which joins the vagus have already been given in the account of the palsy of the laryngeal branches of the pneumogastric. Disease or compression of the external portion is followed by paralysis of the sterno-mastoid and of the trapezius on the same side. In paralysis of one sterno-mastoid, the patient rotates the head with difficulty to the opposite side, but there is no torticollis, though in some cases the head is held obliquely. As the trapezius is supplied in part from the cervical nerves, it is not completely paralyzed, but the portion which passes from the occipital bone to the acromion is functionless. The paralysis of the muscle is well seen when the patient draws a deep breath or shrugs the shoulders. The middle portion of the trapezius is also weakened, the shoulder droops a little, and the angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is impaired, for the trapezius does not fix the scapula as a point from which the deltoid can work.

In progressive muscular atrophy we sometimes see bilateral paralysis of these muscles. Thus, if the sterno-mastoids are affected, the head tends to fall back; when the trapezii are involved, it falls forward, a characteristic attitude of the head in many cases of progressive muscular

atrophy. Gowers suggests that lesions of the accessory in difficult labor may account for those cases in which during the first year of life the child has great difficulty in holding up the head. In children this drooping of the head is an important symptom in cervical meningitis, the result of caries.

The *treatment* of the condition depends much upon the cause. In the central nuclear atrophy but little can be done. In paralysis from pressure the symptoms may gradually be relieved. The paralyzed muscles should be stimulated by electricity and massage.

Accessory Spasm.—(*Torticollis; Wryneck.*)—The forms of spasm affecting the cervical muscles are best considered here, as the muscles supplied by the accessory are chiefly, though not solely, responsible for the condition. The following forms may be described in this section:

(*a*) *Congenital Torticollis.*—This condition, also known as fixed torticollis, depends upon the shortening and atrophy of the sterno-mastoid on one side. It occurs in children and may not be noticed for several years on account of the shortness of the neck, the parents often alleging that it has only recently come on. It affects the right side almost exclusively. A remarkable circumstance in connection with it is the existence of facial asymmetry noted by Wilks, which appears to be an essential part of this congenital form. It occurred in six cases reported by Golding-Bird. In a case recently under my observation, the wryneck was not noticed until her tenth year. The muscle was divided and she seemed quite well; but as she developed the asymmetry of the face became very striking. In congenital wryneck the sterno-mastoid is shortened, hard and firm, and in a condition of more or less advanced atrophy. This must be distinguished from the local thickening in the sterno-mastoid due to rupture, which may occur at the time of birth and produce an induration or muscle callus. Although the sterno-mastoid is almost always affected, there are rare cases in which the fibrous atrophy affects the trapezius. This form of wryneck in itself is unimportant, since it is readily relieved by tenotomy, but Golding-Bird states that the facial asymmetry persists, or indeed may, as shown by photographs in my case, become more evident. With reference to the pathology of the affection, Golding-Bird concludes that the facial asymmetry and the torticollis are integral parts of one affection which has a central origin and is the counterpart in the head and neck of infantile paralysis with talipes in the foot.

(*b*) *Spasmodic Wryneck.*—Two varieties of this spasm occur, the tonic and the clonic, which may alternate in the same case; or, as is most common, they are separate and remain so from the outset. The disease is most frequent in adults and, according to Gowers, most common in females. In this country it is certainly more frequent in males. Of the eight or ten cases which came under my observation in Montreal and Philadelphia, all were males. In females it may be an hysterical manifestation. There may be a marked neurotic family history, but it is usually

impossible to fix upon any definite etiological factor. Some cases have followed cold; others a blow.

The *symptoms* are well defined. In the tonic form the contracted sterno-mastoid draws the occiput toward the shoulder of the affected side; the chin is raised, and the face rotated to the other shoulder. The sterno-mastoid may be affected alone or in association with the trapezius. When the latter is implicated the head is depressed still more toward the same side. In long-standing cases these muscles are prominent and very rigid. There may be some curvature of the spine, the convexity of which is toward the sound side. The cases in which the spasm is clonic are much more distressing and serious. The spasm is rarely limited to a single muscle. The sterno-mastoid is almost always involved and rotates the head so as to approximate the mastoid process to the inner end of the clavicle, turning the face to the opposite side and raising the chin. When with this the trapezius is affected, the depression of the head toward the same side is more marked. The head is drawn somewhat backward; the shoulder, too, is raised by its action. According to Gowers, the splenius is associated with the sterno-mastoid about half as frequently as the trapezius. Its action is to incline the head and rotate it slightly toward the same side. Other muscles may be involved, such as the scalenus and platysma myoides; and in rare cases the head may be rotated by the deep cervical muscles, the rectus and obliquus. There are cases in which the spasm is bilateral, causing a backward movement—the retro-collic spasm. This may be either tonic or clonic, and in extreme cases the face is horizontal and looks upward.

These clonic contractions may come on without warning, or be preceded for a time by irregular pains or stiffness of the neck. The jerking movements recur every few moments, and it is impossible to keep the head still for more than a minute or two. In time the muscles undergo hypertrophy and may be distinctly larger on one side than the other. In some cases the pain is considerable; in others there is simply a feeling of fatigue. The spasms cease during sleep. Emotion, excitement, and fatigue increase them. The spasm may extend from the muscles of the neck and involve those of the face or of the arms.

The disease varies much in its course. Cases occasionally get well, but the great majority of them persist, and, even if temporarily relieved, the disease frequently recurs. The affection is usually regarded as a functional neurosis, but it is possibly due to disturbance of the cortical centres presiding over the muscles.

Treatment.—Temporary relief is sometimes obtained; a permanent cure is exceptional. Various drugs have been used, but rarely with benefit. Occasionally, large doses of bromide will lessen the intensity of the spasm. Morphia, subcutaneously, has been successful in some reported cases, but there is the great danger of establishing the morphia habit. Galvanism may be tried. Counter-irritation is probably

useless. Fixation of the head mechanically can rarely be borne by the patient. These obstinate cases fall ultimately into the hands of the surgeon, and the operations of stretching, division, and excision of the accessory nerve and division of the muscles have been tried. The latter does not check the spasm, and may aggravate the symptoms. Temporary relief may follow, but, as a rule, the condition returns. In the cases of spasm of the deep-seated muscles, Keen has devised an operation for their section.

(*c*) The *nodding spasm* of children may here be mentioned as involving chiefly the muscles innervated by the accessory nerve. It may be a simple trick, a form of habit spasm, or a phenomenon of epilepsy (E. nutans), in which case it is associated with transient loss of consciousness. A similar nodding spasm may occur in older children. In women it sometimes occurs as an hysterical manifestation, commonly as part of the so-called salaam convulsion.

X. Hypoglossal Nerve.

This is the motor nerve of the tongue and for most of the muscles attached to the hyoid bone. Its cortical centre is probably the lower part of the ascending frontal gyrus.

Paralysis.—(1) *Central Lesion.*—The tongue is often paralyzed in hemiplegia, and the paralysis may result from a lesion of the cortex itself, or of the fibres as they pass to the medulla. It does not occur alone and will be considered with hemiplegia. There is this difference, however, between the cortical and other forms, that the muscles on both sides of the tongue may be more or less affected but do not waste, nor are their electrical reactions disturbed.

(2) *Nuclear* and *infra-nuclear* lesions of the hypoglossal result from slow progressive degeneration, as in bulbar paralysis or in locomotor ataxia, and occasionally there is acute softening from obstruction of the vessels. Trauma and lead poisoning have also been assigned as causes. The fibres may be damaged by a tumor, and at the base by meningitis; or the nerve is sometimes involved in its foramen by disease of the skull. The nuclei of both nerves are usually affected together, but may be attacked separately. As a result, there is loss of function in the nerve fibres and the tongue undergoes atrophy on the affected side. It is protruded toward the paralyzed side and may show fibrillary twitching.

The *symptoms* of involvement of one hypoglossal, either at its centre or in its course, are those of unilateral paralysis and atrophy of the tongue. When protruded, it is pushed toward the affected side, and there are fibrillary twitchings. The atrophy is usually marked and the mucous membrane on the affected side is thrown into folds. Articulation is not much impaired in the unilateral affection. When the disease is bilateral, the tongue lies almost motionless in the floor of the mouth; it is atrophied,

and cannot be protruded. Speech and mastication are extremely difficult and deglutition may be impaired. If the seat of the disease is above the nuclei, there may be little or no wasting. The condition is seen in progressive bulbar paralysis and occasionally in progressive muscular atrophy.

The *diagnosis* is readily made and the situation of the lesion can usually be determined, since when supra-nuclear there is associated hemiplegia and no wasting of the muscles of the tongue. Nuclear disease is only occasionally unilateral; most commonly bilateral and part of a bulbar paralysis. It should be borne in mind that the fibres of the hypoglossal may be involved within the medulla after leaving their nuclei. In such a case there may be paralysis of the tongue on one side and paralysis of the limbs on the opposite side, and the tongue, when protruded, is pushed toward the sound side.

Spasm.—This rare affection may be unilateral or bilateral. It is most frequently a part of some other convulsive disorder, such as epilepsy, chorea, or spasm of the facial muscles. In some cases of stuttering, spasm of the tongue precedes the explosive utterance of the words. It may occur in hysteria, and is said to follow reflex irritation in the fifth nerve. The most remarkable cases are those of paroxysmal clonic spasm, in which the tongue is rapidly thrust in and out, as many as forty or fifty times a minute. In the case reported by Gowers the attacks occurred during sleep and continued for a year and a half. The spasm is usually bilateral. Wendt has reported a case in which it was unilateral. The prognosis is usually good.

IV. DISEASES OF THE SPINAL NERVES.

Cervical Plexus.

(1) **Occipito-cervical Neuralgia.**—This involves the nerve territory supplied by the second, the occipitalis major and minor, and the auricularis magnus nerves. The pains are chiefly in the back of the head and neck and in the ear. The condition may follow cold and is sometimes associated with stiffness of the neck or torticollis. Unless connected with disease of the bones or due to pressure of tumors, the outlook is usually good. There are tender points midway between the mastoid process and the spine and just above the parietal eminence, and between the sterno-mastoid and the trapezius. The affection may be due to direct pressure, in persons who carry very heavy loads on the neck.

(2) **Affections of the Phrenic Nerve.**—Paralysis may follow a lesion in the anterior horns at the level of the third and fourth cervical nerves, or may be due to compression of the nerve by tumors or aneurism. More rarely paralysis results from neuritis.

It may be part of a diphtheritic or lead palsy and is usually bilateral.

When the diaphragm is paralyzed respiration is carried on by the intercostal and accessory muscles. When the patient is quiet and at rest little may be noticed, but the abdomen retracts in inspiration and is forced out in expiration. On exertion or even on attempting to move there may be dyspnœa. If the paralysis sets in suddenly there may be dypnœa and lividity, which is usually temporary (W. Pasteur). Intercurrent attacks of bronchitis seriously aggravate the condition. Difficulty in coughing, owing to the impossibility of drawing a full breath, adds greatly to the danger of this complication, as the mucus accumulates in the tubes.

When the phrenic nerve is paralyzed on one side the paralysis may be scarcely noticeable, but careful inspection shows that the descent of the diaphragm is much less on the affected side.

The *diagnosis* of paralysis is not always easy, particularly in women, who habitually use this muscle less than men, and in whom the diaphragmatic breathing is less conspicuous. Immobility of the diaphragm is not uncommon, particularly in diaphragmatic pleurisy, in large effusions, and in extensive emphysema. The muscle itself may be degenerated and its power impaired.

Owing to the lessened action of the diaphragm, there is a tendency to accumulation of blood at the bases of the lungs, and there may be impaired resonance and signs of œdema. As a rule, however, the paralysis is not confined to this muscle, but is part of a general neuritis or an anterior polio-myelitis, and there are other symptoms of value in determining its presence. The outlook is usually serious. Pasteur states that of fifteen cases following diphtheria, only eight recovered. The treatment is that of the neuritis or polio-myelitis with which it is associated.

Brachial Plexus.

(1) **Combined Paralysis.**—The plexus may be involved in the supraclavicular region by compression of the nerve trunks as they leave the spine, or by tumors and other morbid processes in the neck. Below the clavicle lesions are more common and result from injuries following dislocation or fracture, sometimes from neuritis. The most common cause of lesion of the brachial plexus is luxation of the humerus, particularly the subcoracoid form. If the dislocation is quickly reduced the symptoms are quite transient, and disappear in a few days. In severe cases all the branches of the plexus, or only one or two, may be involved. The most serious cases are those in which the dislocation is undetected or unreduced for some time, when the prolonged pressure on the nerves may cause complete and permanent paralysis of the arm. The muscles waste, the reaction of degeneration is present, and trophic changes in the skin are apt to occur. The medico-legal bearings of these cases are important, and may be thus briefly summarized: Direct injury, as by a fall or blow on the shoulder, resulting in great bruising of the nerves without dislocation, is

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occasionally followed by complete paralysis of the arm. A dislocation may be set immediately and yet the lesion of the brachial plexus may be such as to cause permanent paralysis of the nerves. The dislocation may be reduced and the joint in subsequent movements slips out again. It has happened that by the time the surgeon sees the patient again, the damage has become irreparable.

Injuries and blows on the neck may cause partial paralysis of the arm, involving the deltoid, supraspinatus, infraspinatus, biceps, brachialis anticus, and the supinator. The injury may occur to the child during delivery.

A primary neuritis of the brachial plexus is rare. More commonly the process is an ascending neuritis from a lesion of a peripheral branch, involving first the radial or ulnar nerves, and spreading upward to the plexus, producing gradually complete loss of power in the arm.

(2) **Lesions of Individual Nerves of the Plexus.**—(*a*) *Long Thoracic Nerve* (*Serratus Palsy*).—This occurs chiefly in men. The nerve is injured in the posterior triangle of the neck, usually by direct pressure in the carrying of loads; cold may cause neuritis. It may be involved also in progressive muscular atrophy and in polio-myelitis anterior. When paralyzed the scapula on the affected side looks winged, which results from the projection of the angle and posterior border. This is particularly noticeable when the arm is moved forward, when the serratus no longer holds the scapula against the thorax. It is a well-defined and readily recognized form of paralysis. The onset is associated with, sometimes preceded by, neuralgic pains. The course is dubious, and many months may elapse before there is any improvement.

(*b*) *Circumflex Nerve.*—This supplies the deltoid and the teres minor. The nerve is apt to be involved in injuries, in dislocations, bruising by a crutch, or sometimes by extension of inflammation from the joint. Occasionally the paralysis arises from a pressure neuritis during an illness. As a consequence of loss of power in the deltoid, the arm cannot be raised. The wasting is usually marked and changes the shape of the shoulder. Sensation may also be impaired in the skin over the muscle. The joint may be relaxed and there may be a distinct space between the head of the humerus and the acromion. In other instances the ligaments are thickened, and a condition not unlike ankylosis may be produced, which is readily distinguished on moving the arm.

(*c*) *Musculo-spiral Paralysis; Radial Paralysis.*—This is one of the most common of peripheral palsies, and results from the exposed position of the musculo-spiral nerve. It is often bruised in the use of the crutch, by injuries of the arm, blows, or fractures. It is frequently injured when a person falls asleep with the arm over the back of a chair, or by pressure of the body upon the arm when a person is sleeping on a bench or on the ground. It may be paralyzed by sudden violent contraction of the triceps. It is sometimes involved in a neuritis from cold, but this is uncommon in

comparison with other causes. In the subcutaneous injection of ether the nerve may be accidentally struck and temporarily paralyzed. The paralysis of lead poisoning is the result of involvement of certain branches of this nerve.

A lesion when high up involves the triceps, the brachialis anticus, and the supinator longus, as well as the extensors of the wrist and fingers. Naturally, in lesions just above the elbow the arm muscles and the supinator longus are spared. The most characteristic feature of the paralysis is the wrist-drop and the inability to extend the first phalanges of the fingers and thumb. In the pressure palsies the supinators are usually involved and the movements of supination cannot be accomplished. The sensations may be impaired, or there may be marked tingling, but the loss of sensation is rarely so pronounced as that of motion.

The affection is readily recognized, but it is sometimes difficult to say upon what it depends. The sleep and pressure palsies are, as a rule, unilateral and involve the supinator longus. The paralysis from lead is bilateral and the supinators are unaffected. Bilateral wrist-drop is a very common symptom in many forms of multiple neuritis, particularly the alcoholic; but the mode of onset and the involvement of the legs and arms are features which make the diagnosis easy. The duration and course of the musculo-spiral paralysis are very variable. The pressure palsies may disappear in a few days. Recovery is the rule, even when the affection lasts for many weeks. The electrical examination is of importance in the prognosis, and the rules laid down under paralysis of the facial nerve hold good here.

The treatment is that of neuritis.

(*d*) *Ulnar Nerve.*—The motor branches supply the ulnar halves of the deep flexor of the fingers, the muscles of the little finger, the interossei, the adductor and the inner head of the short flexor of the thumb, and the ulnar flexor of the wrist. The sensory branches supply the ulnar side of the hand—two and a half fingers on the back, and one and a half fingers on the front. Paralysis may result from pressure, usually at the elbow-joint, although the nerve is here protected. Possibly the neuritis in the ulnar nerve in some cases of acute illness may be due to this cause. Gowers mentions the case of a lady who twice had ulnar neuritis after confinement. Owing to paralysis of the ulnar flexor of the wrist, the hand moves toward the radial side; adduction of the thumb is impossible; the first phalanges cannot be flexed, and the others cannot be extended. In long-standing cases the first phalanges are overextended and the others strongly flexed, producing the claw-hand; but this is not so marked as in the progressive muscular atrophy. The loss of sensation corresponds to the sensory distribution just mentioned.

(*e*) *Median Nerve.*—This supplies the flexors of the fingers except the ulnar half of the deep flexors, the abductor and the flexors of the thumb, the two radial lumbricales, the pronators, and the radial flexor of the wrist.

The sensory fibres supply the radial side of the palm and the front of the thumb, the first two fingers and half the third finger, and the dorsal surfaces of the same three fingers.

This nerve is seldom involved alone. Paralysis results from injury and occasionally from neuritis. The signs are inability to pronate the forearm beyond the mid-position. The wrist can only be flexed toward the ulnar side; the thumb cannot be opposed to the tips of fingers. The second phalanges cannot be flexed on the first; the distal phalanges of the first and second fingers cannot be flexed; but in the third and fourth fingers this action can be performed by the ulnar half of the flexor profundus. The loss of sensation is in the region corresponding to the sensory distribution already mentioned. The wasting of the thumb muscles, which is usually marked in this paralysis, gives to it a characteristic appearance.

Lumbar and Sacral Plexuses.

The *lumbar plexus* is sometimes involved in growths of the lymph glands, in psoas abscess, and in disease of the bones of the vertebræ. Of its branches the *obturator nerve* is occasionally injured during parturition When paralyzed the power is lost over the adductors of the thigh and one leg cannot be crossed over the other. Outward rotation is also disturbed. The *anterior crural nerve* is sometimes involved in wounds or in dislocation of the hip-joint, less commonly during parturition, and sometimes by disease of the bones and in psoas abscess. The special symptoms of affection of this nerve are paralysis of the extensors of the knee with wasting of the muscles, anæsthesia of the antero-lateral parts of thigh and of the inner side of the leg to the big toe. This nerve is sometimes involved early in growths about the spine, and there may be pain in its area of distribution. Loss of the power of abducting the thigh results from paralysis of the *gluteal nerve*, which is distributed to the gluteus, medius, and minimus muscles.

The *sacral plexus* is frequently involved in tumors and inflammations within the pelvis and may be injured during parturition. Neuritis is common, usually an extension from the sciatic nerve.

Of the branches, the *sciatic nerve*, when injured at or near the notch, causes paralysis of the flexors of the legs and the muscles below the knee, but injury below the middle of the thigh involves only the latter muscles. There is also anæsthesia of the outer half of the leg, the sole, and the greater portion of the dorsum of the foot. Wasting of the muscles frequently follows, and there may be trophic disturbances. In paralysis of one sciatic the leg is fixed at the knee by the action of the quadriceps extensors and the patient is able to walk.

Paralysis of the *small sciatic nerve* is rarely seen. The gluteus maximus is involved and there may be difficulty in rising from a seat. There is a strip of anæsthesia along the back of the middle third of the thigh.

External Popliteal Nerve.—Paralysis involves the peronæi, the long extensors of the toes, tibialis anticus, and the extensor brevis digitorum. The ankle cannot be flexed, resulting in a condition known as foot-drop, and as the toes cannot be raised the whole leg must be lifted, producing the characteristic *steppage* gait seen in so many forms of peripheral neuritis. In long-standing cases the foot is permanently extended and there is wasting of the anterior tibial and peroneal muscles. The loss of sensation is in the outer half of the front of the leg and on the dorsum of the foot.

Internal Popliteal Nerve.—When paralyzed plantar flexion of the foot and flexion of the toes are impossible. The foot cannot be adducted, nor can the patient rise on tiptoe. In long-standing cases talipes calcaneus follows and the toes assume a claw-like position from secondary contracture, due to overextension of the proximal and flexion of the second and third phalanges.

Sciatica.

This is, as a rule, a neuritis either of the sciatic nerve or of its cords of origin. It may in some instances be a functional neurosis or neuralgia.

It occurs most commonly in adult males. A history of rheumatism or of gout is present in many cases. Exposure to cold, particularly after heavy muscular exertion, or a severe wetting are not uncommon causes. Within the pelvis the nerves may be compressed by large ovarian or uterine tumors, by lymphadenomata, by the fœtal head during labor, and occasionally lesions of the hip-joint induce a secondary sciatica. The condition of the nerve has been examined in a few cases, and it has often been seen in the operation of stretching. It is, as a rule, swollen, reddened, and in a condition of interstitial neuritis. The affection may be most intense at the sciatic notch or in the nerve about the middle of the thigh.

Of the *symptoms*, pain is the most constant and troublesome. The onset may be severe, with slight pyrexia, but, as a rule, it is gradual, and for a time there is only slight pain in the back of the thigh, particularly in certain positions or after exertion. Soon the pain becomes more intense, and instead of being limited to the upper portion of the nerve, extends down the thigh, reaching the foot and radiating over the entire distribution of the nerve. The patient can often point out the most sensitive spots, usually at the notch or in the middle of the thigh; and on pressure these are exquisitely painful. The pain is described as gnawing or burning, and is usually constant, but in some instances is paroxysmal, and often worse at night. On walking it may be very great; the knee is bent and the patient treads on the toes, so as to relieve the tension on the nerve. In protracted cases there is wasting of the muscles, but the reaction of degeneration can seldom be obtained. In these chronic cases cramp may occur and fibrillar contractions. Herpes may develop, but this is un-

usual. In rare instances the neuritis ascends and involves the spinal cord.

The duration and course are extremely variable. As a rule it is an obstinate affection, lasting for months, or even, with slight remissions, for years. Relapses are not uncommon, and the disease may be relieved in one nerve only to appear in the other. In the severer forms the patient is bedridden, and such cases prove among the most distressing and trying which the physician is called upon to treat.

In the *diagnosis* it is important, in the first place, to determine whether the disease is primary, or secondary to some affection of the pelvis or of the spinal cord. A careful rectal examination should be made, and, in women, pelvic tumor should be excluded. Lumbago may be confounded with it. Affections of the hip-joint are easily distinguished by the absence of tenderness in the course of the nerve and the sense of pain on movement of the hip-joint or on pressure in the region of the trochanter. There are instances of sacro-iliac disease in which the patient complains of pain in the upper part of the thigh, which may sometimes radiate; but careful examination will readily distinguish between the affections. Pressure on the nerve trunks of the cauda equina, as a rule, causes bilateral pain and disturbances of sensation, and, as double sciatica is rare, these circumstances always suggest lesion of the nerve roots. Between the severe lightning pains of tabes and sciatica the differences are usually well defined.

Treatment.—The pelvic organs should be carefully and systematically examined. Constitutional conditions, such as rheumatism and gout, should receive appropriate treatment. In a few cases with pronounced rheumatic history, which come on acutely with fever, the salicylates seem to do good. In other instances they are quite useless. If there is a suspicion of syphilis the iodide of potassium should be employed, and in gouty cases salines.

Rest in bed with fixation of the limb by means of a long splint is a most valuable method of treatment in many cases, one upon which Weir Mitchell has specially insisted. I have known it to relieve, and in some instances to cure, obstinate and protracted cases which had resisted all other treatment. Hydrotherapy is sometimes satisfactory, particularly the warm baths or the mud baths. Many cases are relieved by a prolonged residence at one of the thermal springs.

Antipyrin, antifebrin, and quinine, are of doubtful benefit.

Local applications are more beneficial. The hot iron or the thermocautery or blisters relieve the pain temporarily. Deep injections into the nerves give great relief and may be necessary for the pain. It is best to use cocaine at first, in doses of from an eighth to a quarter of a grain. If the pain is unbearable morphia may be used, but it is a dangerous remedy in sciatica and should be withheld as long as possible. The disease is so protracted, so liable to relapse, and the patient's *morale* so undermined by

the constant worry and the sleepless nights, that the danger of contracting the morphia habit is very great. On no consideration should the patient be permitted to use the hypodermic needle himself. It is remarkable how promptly, in some cases, the injection of distilled water into the nerve will relieve the pain. Acupuncture may also be tried; the needles should be thrust deeply into the most painful spot for a distance of about two inches, and left for from fifteen to twenty minutes. The injection of chloroform into the nerve has also been recommended.

Electricity is an uncertain remedy. Sometimes it gives prompt relief; in other cases it may be used for weeks without the slightest benefit. It is most serviceable in the chronic cases in which there is wasting of the legs, and should be combined with massage. The galvanic current should be used; a flat electrode should be placed over the sciatic notch, and a smaller one used along the course of the nerve and its branches. In very obstinate cases nerve-stretching may be employed. It is sometimes successful; but in other instances the condition recurs and is as bad as ever.

II. DISEASES OF THE SPINAL CORD.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater.

Pachymeningitis.—The dura mater of the cord is separated by a loose connective tissue from the bony canal in which it lies, and an inflammation may involve either its outer or its inner aspect; hence the division into pachymeningitis externa and interna.

(*a*) *Pachymeningitis Externa.*—This is invariably a secondary inflammation and is occasionally met with in an *acute* form in caries or in syphilitic affections of the bone. Abscess may penetrate the spinal canal or the inflammation may even extend to the peridural tissue in long-standing decubitus. The symptoms are usually those of a compression myelitis.

The *chronic* form of external pachymeningitis, also a secondary affection, is much more common. It is a constant accompaniment of tuberculous disease of the spine and plays a very important part in the production of the symptoms. The affection may be confined to the part in immediate connection with the local disease, but in some cases the subdural space over six or eight vertebræ is occupied by caseous masses. The cord at the site of the curvature in Pott's disease may be compressed, with perhaps little or no involvement of the pia mater. The internal surface of the dura may be perfectly smooth, perhaps a little adherent to the arachnoid, while the external dura is thickened, rough, and covered with a cheesy substance of a variable degree of consistence. In some instances the dura is completely surrounded by this material; in others it is chiefly

Gout may be associated with

on the anterior surface. We can understand the recovery in cases of compression paraplegia if we bear in mind that in large part the actual compression is produced by this material between the diseased vertebræ and the dura mater. The symptoms are those of myelitis from compression, often with signs of involvement of the nerve roots, such as will be mentioned in the next section.

(*b*) *Pachymeningitis interna*, described by Charcot and Joffroy, involves chiefly the cervical region (*P. cervicalis hypertrophica*). The interspace between the cord and the dura is occupied by a firm, concentrically arranged, fibrinous growth, which is seen to have developed within, not outside of, the dura mater. It is a condition anatomically identical with the hæmorrhagic pachymeningitis interna of the brain. The cord is usually compressed; the central canal may be dilated—hydromyelus—and there are secondary degenerations. The nerve roots are involved in the growth and are damaged and compressed. The extent is variable. It may be limited to one segment, but more commonly involves a considerable portion of the cervical enlargement. The disease is chronic, and in some cases presents a characteristic group of symptoms. There are intense neuralgic pains in the course of the nerves whose roots are involved. They are chiefly in the arms and in the cervical region, and vary greatly in intensity. There may be hyperæsthesia with numbness and tingling; atrophic changes may develop, and there may be areas of anæsthesia. Gradually motor disturbances appear; the arms become weak and the muscles atrophied, particularly in certain groups, as the flexors of the hand. The extensors, on the other hand, remain intact, so that the condition of claw-hand is gradually produced. The grade of the atrophy depends much upon the extent of involvement of the cervical nerve roots, and in many cases the atrophy of the muscles of the shoulders and arms becomes extreme. The condition is one of cervical paraplegia, with contractures, flexion of the wrist, and typical *main en griffe*. Usually before the arms are greatly atrophied there are the symptoms of what the French writers term the second stage—namely, involvement of the lower extremities and the gradual production of a spastic paraplegia, which may develop several months after the onset of the disease, and is due to secondary changes in the cord.

The disease runs a chronic course, lasting, perhaps, two or more years. In a few instances, in which symptoms pointed definitely to this condition, recovery has taken place. The disease is to be distinguished from amyotrophic lateral sclerosis, syringomyelia, and tumors. From the first it is separated by the marked severity of the initial pains in the neck and arms; from the second, by the absence of the sensory changes characteristic of syringomyelia. From certain tumors it is very difficult to distinguish, as, in fact, the fibrinous layers form a tumor around the cord.

The condition known as *hæmatoma* of the dura mater may occur at any part of the cord, or, in its slow, progressive form—pachymeningitis

hæmorrhagica interna—may be limited to the cervical region and produce the symptoms just mentioned. It is sometimes extensive, and may coexist with a similar condition of the cerebral dura. Cysts may occur filled with hæmorrhagic contents.

Diseases of the Pia Mater.

(*a*) Acute Spinal Meningitis; Leptomeningitis.

Etiology.—Spinal meningitis occurs: (1) In tuberculosis. This is perhaps the most common form in general practice and has already been considered. (2) In specific cerebro-spinal meningitis, which occurs endemically or epidemically, and has also been considered under its appropriate section. (3) As a secondary involvement in certain infectious diseases, pneumonia, small-pox, scarlet fever, and typhoid fever. This form is very rare. Even in pneumonia, in which the cerebral meninges are frequently involved, the spinal meninges are seldom affected, except perhaps in the first two or three inches of the cervical region. (4) From injury or the extension of inflammation, as after operation on spina bifida. (5) There are cases in which the meningitis appears to have followed exposure to cold and wet.

Morbid Anatomy.—The affection may be diffused over the entire cord or localized to the cervical region. In the early stage the vessels of the pia mater are injected. The fluid in the pia-arachnoid space is slightly turbid. In some intense grades, on opening the dura the contour of the cord cannot be seen, as it is completely enveloped in a sero-fibrinous or purulent exudate, which here and there causes bulging of the arachnoid. Owing to the position of the body, the exudate is most abundant in the posterior part, or sinks to the lumbar region. In acute cases the pia itself does not look thickened, but in more chronic forms the membrane may be grayish and turbid. In a majority of instances, if the inflammation is intense, the exudate is seen in the anterior and posterior median fissures and the cortical portion of the cord is swollen and infiltrated, so the condition can be properly called meningomyelitis. The affection may be limited to the spinal meninges, but in a majority of instances it is a cerebro-spinal lesion.

Symptoms.—These have already been referred to in considering the two commonest varieties, the tuberculous and the epidemic. The disease often sets in with a chill and fever. Pain in the back, stiffness in the neck, pain on pressure along the vertebræ, tremor or spasm of the muscles, and disturbances of sensation are usually present. Girdle sensations are not common. The reflexes may be increased. Later, paralytic symptoms may develop, but they are uncommon, except in pure spinal meningitis.

The *diagnosis* is often difficult. In a large proportion of the cases supposed to be spinal meningitis the membranes are not inflamed. I have

already referred to the identity of the spinal symptoms in certain of the infectious diseases with those of acute leptomeningitis. In the case of a patient with high fever, marked stiffness of the back and neck muscles, or opisthotonus with rigidity and tremor of the muscles, it is not unnatural to make a positive diagnosis of spinal meningitis, but every symptom of the condition may be present without any inflammatory exudate. The truth of Stokes's dictum, already quoted (p. 25), has been brought home to me on many occasions. On the other hand, there are instances of well-marked leptomeningitis, more particularly the cerebro-spinal form, in which spinal symptoms are trifling or absent. To distinguish between the different forms of spinal meningitis is sometimes extremely difficult. A correct diagnosis is oftenest made in tuberculous cases, since here the prodromata are well defined and the symptoms indicative of involvement of the cerebral meninges well marked. There are cases in which the spinal meninges bear the brunt of the affection. I have already referred to one case in which the meningitis was thought to be due to traumatism. The coexistence of disease at the apex of the lungs or of local tuberculous lesions elsewhere is of great value.

The diagnosis of the epidemic form has already been considered.

(*b*) **Chronic Leptomeningitis.**—As a primary lesion this is extremely rare. It sometimes follows the prolonged use of alcohol. It occurs in connection with syphilis, trauma, and as a complication of various sclerosis of the spinal cord, either systemic or insular.

Anatomically the condition is characterized by a thickening and turbidity of the pia, often with adhesions to the arachnoid and the dura. The membranes may be stained with blood-pigment. These alterations may occur in localized spots or over extensive areas. The nerve roots may be involved and thickened. The spinal cord itself is rarely affected, though strands of connective tissue may extend into the cortical zone, producing slight sclerosis. The opaque, white, cartilaginous plates which occur so often on the posterior surface of the spinal arachnoid and are sometimes adherent to the pia cause no symptoms and are not to be mistaken for this chronic meningitis.

The *symptoms* of this form are indefinite. Simple thickening of the meninges may produce no signs during life unless the spinal nerve roots are involved. In any case the diagnosis is somewhat doubtful. There are instances in which pain in the back, stiffness of the dorsal muscles, and pains radiating in the nerves of the trunk or in the extremities have been marked. Hyperæsthesia and skin eruptions may be present. When the cord is involved paralytic symptoms may develop. The reflexes are increased. The course is always chronic, lasting for many years.

The treatment is purely symptomatic. Recovery probably never occurs.

Hæmorrhage into the Spinal Membranes; Hæmatorrhachis.

In meningeal apoplexy, as it is called, the blood may be between the dura mater and the spinal canal—extrameningeal hæmorrhage—or within the dura mater—intrameningeal hæmorrhage.

(*a*) *Extrameningeal Hæmorrhage* occurs usually as a result of trauma. The exudation may be extensive without compression of the cord. The blood comes from the large plexuses of veins which surround the dura. The rupture of an aneurism into the spinal canal may produce extensive and rapidly fatal hæmorrhage.

(*b*) *Intrameningeal Hæmorrhage* is rather more common, but is rarely extensive from causes acting directly on the spinal meninges themselves. Scattered hæmorrhages are not unfrequent in the acute infectious fevers, and I have twice, in malignant small-pox, seen much effusion. Bleeding occurs also in death from convulsive disorders, such as epilepsy, tetanus, and strychnia poisoning. The most extensive hæmorrhages occur in cases in which the blood comes from rupture of an aneurism at the base of the brain, either of the basilar or vertebral. In several cases of this kind I have found a large amount of blood in the spinal meninges. In ventricular apoplexy the blood may pass from the fourth ventricle into the spinal meninges. There is a specimen in the medical museum of McGill College of the most extensive intraventricular hæmorrhage, in which the blood passed into the fourth ventricle, and descended beneath the spinal arachnoid for a considerable distance. On the other hand, hæmorrhage into the spinal meninges may possibly ascend into the brain.

The *symptoms* in moderate grades may be slight and indefinite. In the non-traumatic cases the hæmorrhage may either come on suddenly or after a day or two of uneasy sensations along the spine. As a rule, the onset is abrupt, with sharp pain in the back and symptoms of irritation in the course of the nerves. There may be muscular spasms, or paralysis may come on suddenly, either in the legs alone or both in the legs and arms. In some instances the paralysis develops more slowly and is not complete. There is no loss of consciousness, and there are no signs of cerebral disturbance. The clinical picture naturally varies with the site of the hæmorrhage. If in the lumbar region, the legs alone are involved, the reflexes may be abolished, and the action of the bladder and rectum impaired. In the dorsal region there is more or less complete paraplegia, the reflexes are usually retained, and there are signs of disturbance in the thoracic nerves, such as girdle sensations, pains, and sometimes eruption of herpes. In the cervical region the arms as well as the legs may be involved; there may be difficulty in breathing, stiffness of the muscles of the neck, and occasionally pupillary symptoms.

The prognosis depends much upon the cause of the hæmorrhage. Recovery may take place in the traumatic cases, and in those associated with the infectious diseases.

II. AFFECTIONS OF THE BLOOD-VESSELS.

(*a*) **Congestion.**—Apart from actual myelitis, we rarely see post mortem evidences of congestion of the spinal cord, and when we do it is usually limited either to the gray matter or to a definite portion of the organ. There is necessarily, from the posture of the body post mortem, a greater degree of vascularity in the posterior portion of the cord. The white matter is rarely found congested, even when inflamed; in fact, it is remarkable how uniformly pale this portion of the cord is. The gray matter often has a reddish-pink tint, but rarely a deep reddish hue, except when myelitis is present. If we know little anatomically of conditions of congestion of the cord, we know less clinically, for there are no features in any way characteristic of it.

(*b*) **Anæmia.**—So, too, with this state. There may be extreme grades of anæmia of the cord without symptoms. In chlorosis and pernicious anæmia there are rarely symptoms pointing to the cord, and there is no reason to suppose that such sensations as heaviness in the limbs and tingling are especially associated with anæmia.

There are, however, some very interesting facts with reference to the profound anæmia of the cord which follows ligature of the aorta. In experiments made in Welch's laboratory by Herter, it was found that within a few moments after the application of the ligature to the aorta paraplegia came on. Paralysis of the sphincters developed, but less rapidly. Within fourteen days contractures of the limbs set in with atrophy and fibrillar twitchings. Histologically it was shown that within thirty-six hours there were marked changes in the ganglion cells of the anterior horns in the lumbar segments, and later there were signs of a definite myelitis. This condition is of interest in connection with the fact of the rapid development of a paraplegia after profuse hæmorrhage, usually from the stomach or uterus. It may come on at once or at the end of a week or ten days, and is probably due to an anatomical change in the nerve elements similar to that produced in Herter's experiments.

In this connection may be mentioned the interesting observations of Lichtheim upon the degeneration of the posterior columns of the cord in pernicious anæmia, of which he has reported three cases. He regards it as a form of toxic myelitis, due to the altered condition of the blood.

(*c*) **Embolism and Thrombosis.**—Blocking of the spinal arteries by emboli rarely occurs. It may be produced experimentally, and Money found that it was associated with choreiform movements. Thrombosis of the smaller vessels in connection with endarteritis plays an important part in many of the acute and chronic changes in the cord.

(*d*) **Endarteritis.**—It is remarkable how frequently in persons over fifty the arteries of the spinal cord are found sclerotic. The following forms may be met with: (1) A nodular peri-arteritis or endarteritis associated

with syphilis and sometimes with gummata of the meninges; (2) an arteritis obliterans, with great thickening of the intima and narrowing of the lumen of the vessels, involving chiefly the medium and larger-sized arteries. Miliary aneurisms or aneurisms of the larger vessels are rarely found in the spinal cord. In the classical work of Leyden but a single instance of the latter is mentioned.

(*e*) **Hæmorrhage into the Spinal Cord** (*Hæmatomyelia*).—The existence of a primary hæmorrhage into the cord has been denied on the ground that in all instances it is preceded by a condition of softening. A majority of authors, however, admit the existence of a primary form. About forty-two cases are on record, which are collected in the thesis of Hayem * and in the article of Berkeley.† It is more common in males than in females, and at the middle period of life. The cases have followed either cold and exposure or overexertion, and, most frequently of all, traumatism. It occurs also in tetanus and convulsions. Hæmorrhage may be associated with tumors, with syringo-myelia, or with myelitis; it is often difficult to determine whether the case is one of primary hæmorrhage with myelitis, or myelitis with a secondary hæmorrhage.

The *anatomical condition* is very varied. The cord may be enlarged at the site of the hæmorrhage, and occasionally the white substance may be lacerated and blood may escape beneath the meninges. The extravasation is chiefly in the gray matter, and may be limited or focal, or very diffuse, extending a considerable distance in the cord. In a case which occurred at the Montreal General Hospital under Wilkins the hæmorrhage occupied a position opposite the region of the fifth and sixth cervical nerves and on transverse section the cord was occupied by a dark-red clot measuring twelve by five millimetres, around which the white substance formed a thin, ragged wall. The clot could be traced upward as far as the second cervical, and downward as far as the fourth dorsal.

The sudden onset of the *symptoms* is the most characteristic feature in hæmatomyelia. The loss of power necessarily varies with the locality affected. If in the cervical region, both arms and legs may be involved; but if in the dorsal or lumbar, there is only paraplegia. There is usually loss of sensation, and at first loss of reflexes. Myelitis frequently develops and becomes extensive, with fever and trophic changes. The condition may rapidly prove fatal; in other instances there is gradual recovery, often with partial paralysis.

The diagnosis may be made in some instances, particularly those in which the onset is sudden after injury, but there is great difficulty in differentiating hæmorrhagic myelitis from certain cases of hæmorrhage into the spinal meninges. The question of diagnosis has been carefully considered by Hoch ‡ in a recent report of two cases from my clinic.

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* Paris, 1872.

† Brain, 1889.

‡ Johns Hopkins Hospital Reports, vol. ii, fasciculus 6.

(*f*) **Caisson Disease; Diver's Paralysis.**—This remarkable affection, found in divers and in workers in caissons, is characterized by a paraplegia, more rarely a general palsy, which supervenes on returning from the compressed atmosphere to the surface.

The disease has been carefully studied by the French writers, by Leyden and Schultze in Germany, and in this country particularly by A. H. Smith. The pressure must be more than that of three atmospheres. The symptoms are especially apt to come on if the change from the high to the ordinary atmospheric pressure is quickly made. They may supervene immediately on leaving the caisson, or they may be delayed for several hours. In the mildest form there are simply pains about the knees and in the legs, often of great severity, and occurring in paroxysms. Abdominal pain and vomiting are not uncommon. The legs may be tender to the touch, and the patient may walk with a stiff gait. Dizziness and headache may accompany these neuralgic symptoms, or may occur alone. More commonly in the severe form there is paralysis both of motion and sensation, usually a paraplegia, but it may be general, involving the trunk and arms. Monoplegia and hemiplegia are rare. In the most extreme instances the attacks resemble apoplexy, and the patient rapidly becomes comatose and death occurs in a few hours. In the cases of paraplegia the outlook is usually good, and the paralysis may pass off in a day, or may continue for several weeks or even for months. Identical features are met with in the deep-sea divers.

The explanation of this condition is by no means satisfactory. Two careful autopsies have been made. In Leyden's case death occurred on the fifteenth day, and in the dorsal portion of the cord there were numerous foci of hæmorrhages and signs of an acute myelitis. In Schultze's case death occurred in two and a half months, and a disseminated myelitis was found in the dorsal region. In both cases there were fissures, and appearances as if tissue had been lacerated. It has been suggested that the symptoms are due to the liberation in the spinal cord of bubbles of nitrogen which have been absorbed by the blood under the high pressure, and the condition found at the autopsies just referred to is held to favor this view.

A large majority of the cases recover. The severe neuralgic pains often require morphia. Inhalations of oxygen and the use of compressed air have been advised. When paraplegia develops the treatment is similar to that of other forms. In all caisson work care should be exercised that the time in passing through the lock from the high to the ordinary pressure be sufficiently prolonged. According to A. H. Smith, at least five minutes should be allowed for each additional atmosphere of pressure.

In a case examined on the 3rd day (Zeitschr. Heilkunde 1882) the condition of fissuring & laceration was found.

III. ACUTE AFFECTIONS OF THE SPINAL CORD.

(1) Acute Diffuse Myelitis.

Etiology.—Acute myelitis results from many causes, and may affect the cord in a limited or extended portion—the gray matter chiefly, or the gray and white matter together. It is met with: (*a*) As an independent affection following exposure to cold, or exertion, and leading to rapid loss of power with the symptoms of an acute ascending paralysis. (*b*) As a sequel of the infectious diseases, such as small-pox, typhus, and measles. (*c*) As a result of traumatism, either fracture of the spine or very severe muscular effort. Concussion without fracture may produce it, but this is rare. Acute myelitis, for instance, scarcely ever follows railway accidents. (*d*) In disease of the bones of the spine, either caries or cancer. This is a more common cause of localized acute transverse myelitis than of the diffuse affection. (*e*) In disease of the cord itself, such as tumors and syphilis; in the latter, either in association with gummata, in which case it is usually a late manifestation, or it may follow within a year or eighteen months of the primary affection.*

Morbid Anatomy.—In localized acute myelitis affecting white and gray matter, as met with after accident or an acute compression, the cord is swollen, the pia injected, the consistence greatly reduced, and on incising the membrane an almost diffluent fluid may escape. In less intense grades, on section at the affected area, all trace of distinction between the gray and white matter is lost, or extremely indistinct. The tissue may be injected, or, as is often the case, hæmorrhagic. It is particularly in these forms, due to extension of disease from without or to acute compression, that we find definite involvement of the white matter. In other instances the gray matter is chiefly affected. There may be localized areas throughout the cord in which the gray matter is reduced in consistence and hæmorrhagic, the so-called red softening. There may be definite cavity formations in these foci. In some cases of disseminated or focal myelitis the meninges also are involved and there is a myelo-meningitis. And, lastly, there are instances in which, throughout a long section of the cord, sometimes through the lumbar and the greater part of the dorsal, or in the dorsal and cervical regions, there is a diffuse myelitis of the gray substance.

Histologically the nerve fibres are much swollen and irregularly distorted, the axis cylinders are beaded, the myelin droplets are abundant, and the laminated bodies known as corpora amylacea may be seen. The granular fatty cells are also numerous and there may be leucocytes and red blood-corpuscles. Changes in the blood-vessels are striking; the smaller veins are distended and may show varicosities. The perivascular

* Breteau, Des Maladies Syphilitiques Précoces, Paris Thesis, 1889.

lymph spaces contain numerous leucocytes, and the smaller arteries themselves are frequently the seat of hyaline thrombi. The ganglion cells are swollen and irregular in outline, the protoplasm is extremely granular and vacuolated, and the nuclei, though usually invisible, may show signs of division, and the processes of the cells are not seen.

In cases which persist for some time we have an opportunity of seeing the later stages of acute myelitis. The acute, inflammatory, hyperæmic or red softening is succeeded by stages in which the affected area becomes more yellow from gradual alteration of the blood-pigment, and finally white in color from the advancing fatty degeneration. In cases of compression myelitis, a sclerosis may gradually be produced with the anatomical picture of a chronic diffuse myelitis.

Symptoms.—(*a*) *Acute Central Myelitis.*—It is this form which comes on spontaneously after cold, or in connection with syphilis or one of the infectious diseases, or is seen in a typical manner in the extension from injuries or from tumor. The onset, though scarcely so abrupt as in hæmorrhage, may be sudden; a person may be attacked on the street and have difficulty in getting home. In some instances, the onset is preceded by pains in the legs or back, or a girdle sensation is present. It may be marked by chills, occasionally by convulsions; fever is usually present from the beginning—at first slight, but subsequently it may become high.

The *motor* functions are rapidly lost, sometimes as quickly as in Landry's ascending paralysis. The paraplegia may be complete, and, if the myelitis extends to the cervical region, there may be impairment of motion, and ultimately complete loss of power of the upper extremities as well. The sensation is lost, but there may at first be hyperæsthesia. The reflexes in the initial stage are increased, but in acute central myelitis, unless limited in extent to the dorsal and cervical regions, the reflexes are usually abolished. The rectum and bladder are paralyzed. Trophic disturbances are marked; the muscles waste rapidly; the skin is often congested, and there may be localized sweating. The temperature of the affected limbs may be lowered. Acute bed-sores may develop over the sacrum or on the heels, and sometimes a multiple arthritis is present. In these acute cases the general symptoms become greatly aggravated, the pulse is rapid, the tongue becomes dry; there is delirium, the fever increases, and may reach 107° or 108°.

The course of the disease is variable. In very acute cases death follows in from five to ten days. The cases following the infectious diseases particularly the fevers and sometimes syphilis, may run a milder course.

The diagnosis of this variety of acute myelitis is rarely difficult. In common with the acute ascending paralysis of Landry, and with certain cases of multiple neuritis, it presents a rapid and progressive motor paralysis. From the former it is distinguished by the more marked involvement of sensation, the trophic disturbances, the paralysis of bladder and rectum,

the rapid wasting, the electrical changes, and the fever. From acute cases of multiple neuritis it may be more difficult to distinguish, as the sensory features in these cases may be marked, though there is rarely, if ever, in multiple neuritis complete anæsthesia; the wasting, moreover, is more rapid in myelitis. The bladder and rectum are rarely involved—though in exceptional cases they may be—and, most important of all, the trophic changes, the development of bullæ, bed-sores, etc., are not seen in multiple neuritis.

(*b*) *Acute Transverse Myelitis.*—The symptoms naturally differ with the situation of the lesion.

(1) Acute transverse myelitis in the *dorsal region*, the most common situation, produces a very characteristic picture. The symptoms of onset are variable. There may be initial pains or numbness and tingling in the legs. The paralysis may set in quickly and become complete within a few days; but more commonly it is preceded for a day or two by sensations of pain, heaviness, and dragging in the legs. The paralysis of the lower limbs is usually complete, and if at the level, say, of the sixth dorsal vertebra, the abdominal muscles are involved. Sensation may be partially or completely lost. At the onset there may be numbness, tingling, or even hyperæsthesia in the legs. At the level of the lesion there is often a zone of hyperæsthesia, which is discovered by passing a test-tube containing hot water along the spine, when the sensation of warmth changes to one of actual pain. A girdle sensation may occur early, and when the lesion is in this situation it is usually felt between the ensiform and umbilical regions. The reflex functions are variable. There may at first be abolition of the reflexes; subsequently, the reflexes, passing through the segments lower than the one affected, may be exaggerated and the limbs may pass into a condition of spastic rigidity. It does not always happen, however, that the reflexes are increased in a total transverse lesion of the cord. They may be entirely lost, as pointed out some years ago by Bastian, and insisted upon by him in a recent memoir.* F. T. Miles has also called attention to this fact and reported five cases in which the reflexes were lost in total transverse lesion of the cord. That this is not due to the preliminary shock is shown by the fact that the abolition of the reflexes may continue for four or more months. The trophic changes are not marked. The muscles become extremely flabby, but not wasted in an extreme degree; subsequently rigidity develops. If the gray matter of the lumbar cord is involved, the flaccidity persists and the wasting may be considerable. The reaction of degeneration is not present. The temperature of the paralyzed limbs is variable. It may at first rise, then fall and become subnormal. Lesions of the skin are not uncommon, and bed-sores are apt to form. There is at first retention of urine and subsequent incontinence. If the lumbar centres are involved, there are from the outset vesical symptoms. The urine is alka-

* Medico-Chirurgical Transactions, vol. lxxiii.

line in reaction and may rapidly become ammoniacal. The bowels are constipated and there is usually incontinence of the fæces. Some writers attribute the cystitis associated with transverse myelitis to disturbed trophic influence.

The course of complete transverse myelitis depends a good deal upon its cause. Death may result from extension. Segments of the cord may be completely and permanently destroyed, in which case there is persistent paraplegia. The pyramidal fibres below the lesion undergo the secondary degeneration, and there is an ascending degeneration of the posterior median columns. If the lower segments of the cord are involved the legs may remain flaccid. In some instances a transverse myelitis of the dorsal region involves the anterior horns above and below the lesion, producing flaccidity of the muscles, with wasting, fibrillar contractions, and the reaction of degeneration. More commonly, however, in the cases which last many months there is more or less rigidity of the muscles with spasm or persistent contraction of the flexors of the knee.

(2) *Transverse Myelitis of the Cervical Region.*—If at the level of the sixth or seventh cervical nerves, there is paralysis of the upper extremities, more or less complete, sometimes sparing the muscles of the shoulder. Gradually there is loss of sensation. The paralysis is usually complete below the point of lesion, but there are rare instances in which the arms only are affected, the so-called cervical paraplegia. In addition to the symptoms already mentioned there are several which are more characteristic of transverse myelitis in the cervical region, such as the occurrence of vomiting, hiccough, and slow pulse, which may sink to twenty or thirty, pupillary changes—myosis—sometimes attacks of dysphagia, dyspnœa, or syncope.

II. Myelitis of the Anterior Horns

(*Polio-myelitis Anterior; Atrophic Spinal Paralysis*).

Definition.—An affection occurring most commonly within the first three years of life, characterized by fever, loss of power in certain muscles, and rapid atrophy.

Etiology.—The cause of the disease is unknown. It has been attributed to cold, to the irritation from dentition, or to overexertion. Since the days of Mephibosheth, parents have been inclined to attribute this form of paralysis to the carelessness of nurses in letting the children fall, but very rarely is the disease induced by traumatism, and in perhaps a majority of the cases the child is attacked while in full health. As Sinkler has pointed out, the cases are more common in the warm months. Boys are more liable to be affected than girls. Several instances of the occurrence of numerous cases together in epidemic form have been described. Medin reports from Stockholm an epidemic in which from the 9th of August to the 23d of September 29 cases came under observation. In two instances two children in the same family were attacked within a few days.

Although most frequent in children, it develops occasionally in young adults, or even in middle-aged persons.

Morbid Anatomy.—The disease is oftenest seen in either the cervical or lumbar enlargements. In very early cases, such as those described by David Drummond and Charlewood Turner, the lesion has been that of an acute hæmorrhagic myelitis with degeneration and rapid destruction of the large ganglion cells. The condition may be strictly confined to the anterior cornua; in some instances there is slight meningeal involvement. In cases in which the examination is not made for some months or years the changes are very characteristic. The anterior cornu in the affected region is greatly atrophied and the large motor cells are either entirely absent or only a few remain. The affected half of the cord may be considerably smaller than the other. The antero-lateral column may show slight sclerotic changes, chiefly in the pyramidal tract. The corresponding anterior nerve roots are atrophied, and the muscles are wasted and gradually undergo a fatty and sclerotic change.

Symptoms.—In a majority of the cases, after slight indisposition and feverishness, the child is noticed to have lost the use of one limb. Convulsions at the outset are rare, not constant as in the acute cerebral palsies of children. Fever is usually present, the temperature rising to 101°, sometimes to 103°. Pain is rarely complained of; there may occasionally be slight aching in the joints. The paralysis is abrupt in its onset and, as a rule, is not progressive, but reaches its maximum in a very short time, even within twenty-four hours. It is rarely generalized. The suddenness of onset is remarkable and suggests a primary affection of the blood-vessels, a view which the hæmorrhagic character of the early lesion supports. The distribution of the paralysis is very variable. One or both arms may be affected, one arm and one leg, or both legs; or it may be crossed paralysis, the right leg with the left arm. In the upper extremities the paralysis is rarely complete and groups of muscles may be affected. As Remak has pointed out, there is an upper-arm and a lower-arm type of palsy. The deltoid, the biceps, brachialis anticus, and supinator longus may be affected in the former, and in the latter the extensors or flexors of the fingers and wrists. This distribution is due to the fact that the groups of nerve-cells are attacked which preside over certain muscles acting functionally together.

In the legs the tibialis anticus and extensor groups of muscles are more affected than the hamstrings and glutei. The muscles of the face are ~~never~~, the sphincters ~~rarely~~, involved. While the rule is for the paralysis to be abrupt and sudden, there are cases in which it comes on slowly and takes from three to five days for its development. At first the affected limb looks natural, and as children between two and three are usually fat, very little change may be noticed for some time; but the atrophy proceeds rapidly, and the limb becomes flaccid and feels soft and flabby. Usually as early as the end of the first week the reaction of degeneration

very rarely, if hardly ever

is present. The nerves are found to have lost their irritability. The muscles do not react to the induced current, but to the constant current they respond by a sluggish contraction, usually to a weaker current than is normal, and more to the positive pole than to the negative. The paralysis remains stationary for a time, and then there is gradual improvement. Complete recovery is rare, and, when the anatomical condition is considered, is scarcely to be expected. The large motor cells of the cornua, when thoroughly disintegrated, cannot be restored. In too many cases the improvement is only slight and permanent paralysis remains in certain groups. Sensation is unaffected; the skin reflexes are absent, and the deep reflexes are usually lost.

When the paralysis persists the wasting is extreme, the growth of the bones of the affected limb is arrested, or at any rate retarded, and the joints may be very relaxed; as, for instance, when the deltoid is affected the head of the humerus is no longer kept in contact with the glenoid cavity. In the later stages very serious deformities are produced by the contracture of the muscles.

Diagnosis.—The condition is only too evident in the majority of cases. There is a flaccid, flabby paralysis of one or more limbs which has set in abruptly. The rapid wasting, the lax state of the muscles, the electrical reactions, and the absence of reflexes distinguish it from the cerebral palsies. The pseudo-paresis of rickets is a condition to be carefully distinguished. In this the loss of power is in the legs, rapid atrophy is not present, certain movements are possible but painful. The general hyperæsthesia of the skin, the characteristic changes in the bones, and the diffuse sweats are present. Disease of the hip or knee may produce a pseudo-paralysis which can with care be readily distinguished.

Prognosis.—The outlook in any case for complete recovery is bad. The natural course of the disease must be borne in mind; the sudden onset, the rapid but not progressive loss of power, a stationary period, then marked improvement in certain muscle groups, and finally in many cases contractures and deformities. There is no other disease in which the physician is so often subject to unjust criticism, and the friends should be told at the outset that in the severe and extensive paralysis complete recovery should not be expected. The best to be hoped for is a gradual restoration of power in certain muscle groups. In estimating the probable grade of permanent paralysis, the electrical examination is of great value.

Treatment of Acute Myelitis.—In the rapidly developing form due either to a diffuse inflammation in the gray matter or to transverse myelitis, the important measures are: Scrupulous cleanliness, care and watchfulness in guarding against bed-sores, the avoidance of cystitis, either by systematic catheterization or, if there is incontinence, by a carefully adjusted bed urinal, or the use of antiseptic cotton-wool repeatedly changed. In an acute onset in a healthy subject the spine may be cupped. Counter-irritation is of doubtful advantage. Chapman's ice-bag is some-

times useful. No drugs have the slightest influence upon an acute myelitis, and even in subjects with well-marked syphilis neither mercury nor iodide of potassium is curative. Tonic remedies, such as quinine, arsenic, and strychnia, may be used in the later stages. When the muscles have wasted, massage is beneficial in maintaining their nutrition. Electricity should not be used in the early stages of myelitis. It is of no value in the transverse myelitis in the dorsal region with retention of the nutrition in the muscles of the leg.

The treatment of acute infantile paralysis has a bright and a dark side. In a case of any extent complete recovery cannot be expected; on the other hand, it is remarkable how much improvement may finally take place in a limb which is at first completely flaccid and helpless. The following treatment may be pursued: If seen in the febrile stage, a brisk laxative and a fever mixture may be given. The child should be in bed and the affected limb or limbs wrapped in cotton. As in the great majority of cases the damage is already done when the physician is called and the disease makes no further progress, the application of blisters and other forms of counter-irritation to the back is irrational and only cruel to the child.

The general nutrition should be carefully maintained by feeding the child well, and taking it out of doors every day. As soon as the child can bear friction the affected part should be carefully rubbed; at first once a day, subsequently morning and evening. Any intelligent mother can be taught systematically to rub, knead, and pinch the muscles, using either the bare hand or, better still, sweet oil or cod-liver oil. This is worth all the other measures advised in the disease, and should be systematically practised for months, or even, if necessary, a year or more. Electricity has a much more limited use, and cannot be compared with massage in maintaining the nutrition of the muscles. The faradic current should be applied to those muscles which respond. The essence of the treatment is in maintaining the nutrition of the muscles, so that in the gradual improvement which takes place in parts, at least, of the affected segments of the cord the motor impulses may have to deal with well-nourished, not atrophied muscle fibres.

Of medicines, in the early stage ergot and belladonna have been warmly recommended, but it is unlikely that they have the slightest influence. Later in the disease strychnia may be used with advantage in one or two minim doses of the liquor strychninæ, which, if it has no other effect, is a useful tonic.

The most distressing cases are those which come under the notice of the physician six, eight, or twelve months after the onset of the paralysis, when one leg or one arm or both legs are flaccid and have little or no motion. Can nothing be done? A careful electrical test should be made to ascertain which muscles respond. This may not be apparent at first, and several applications may be necessary before any contractility is

noticed. With a few lessons an intelligent mother can be taught to use the electricity as well as to apply the massage. If in a case in which the paralysis has lasted for six or eight months no observable improvement takes place in the next six months with thorough and systematic treatment, little or no hope can be entertained of further change.

In the later stage care should be taken to prevent the deformities resulting from the contractions. Great benefit results from a carefully applied apparatus.

III. Acute and Subacute Polio-myelitis in Adults.

An acute polio-myelitis in adults, the exact counterpart of the disease in children, is recognized. A majority, however, of the cases described under this heading have been multiple neuritis; but the suddenness of onset, the rapid wasting, and the marked reaction of degeneration are thought by some to be distinguishing features. Multiple neuritis may, however, set in with rapidity; there may be great wasting and the reaction of degeneration is sometimes present. The time element alone may determine the true nature. Recovery in a case of extensive multiple paralysis from polio-myelitis will certainly be with loss of power in certain groups of muscles; whereas, in multiple neuritis the recovery, while slow, may be perfect.

The subacute form, the *paralysie générale spinale antérieure subaiguë* of Duchenne, is in all probability a peripheral palsy. The paralysis usually begins in the legs with atrophy of the muscles, then the arms are involved, but not the face. Sensation is, as a rule, not involved.

IV. Acute Ascending (Landry's) Paralysis.

Definition.—An advancing paralysis, beginning in the legs, rapidly extending to the trunk and arms, and finally, in many cases, involving the muscles of respiration. It presents a remarkable similarity in its symptoms to certain cases of polyneuritis, with which it is now grouped by many writers.

Etiology and Pathology.—The disease occurs most commonly in males between the twentieth and thirtieth years. It has sometimes followed the specific fevers. An elaborate study of 93 cases collected from the literature has been made by James Ross, who concludes that in etiology, symptoms, course, and termination it conforms to a peripheral neuritis. Neuwerk and Barth have reached a similar conclusion. In their case an interstitial neuritis was found in the nerve roots, but the peripheral nerves were normal. On the other hand, cases have been reported of rapidly ascending paralysis in which the periphral nerves and nerve roots were unaffected. In a case of eleven days' duration recently studied by Hun, the lesions were certainly too slight to account for the advancing and wide-spread paralysis, and, with our present knowledge, Hun is cor-

rect in stating that "acute ascending paralysis—defined so as to exclude all cases in which the sensory symptoms are prominent, or in which well-marked bulbar symptoms are not present—must therefore be regarded as a clinical entity for which no corresponding lesion has as yet been discovered." It is not improbable that some toxic agent is responsible for the symptoms.

Symptoms.—Weakness of the legs, gradually progressing, often with tolerable rapidity, is the first symptom. In some cases within a few hours the paralysis of the legs becomes complete. The muscles of the trunk are next affected, and within a few days, or even less in more acute cases, the arms are also involved. The neck muscles are next attacked, and finally the muscles of respiration, deglutition, and articulation. The reflexes are lost, but the muscles neither waste nor show electrical changes. The sensory symptoms are variable; in some cases tingling, numbness, and hyperæsthesia have been present. In the more characteristic cases sensation is intact and the sphincters are uninvolved. Enlargement of the spleen has been noticed in several cases. The course of the disease is variable. It may prove fatal in less than two days. Other cases persist for a week or for two weeks. In some instances recovery has occurred, but in a large proportion of the cases the disease is fatal.

The *diagnosis* is difficult, particularly from certain forms of multiple neuritis, and if we include in Landry's paralysis the cases in which sensation is involved, distinction between the two affections is impossible. We apparently have to recognize the existence of a rapidly advancing motor paralysis without involvement of the sphincters, without wasting or electrical changes in the muscles, without trophic lesions, and without fever—features sufficient to distinguish it from either the acute central myelitis or the polio-myelitis anterior. It is doubtful, however, whether these characters always suffice to enable us to differentiate the cases of multiple neuritis.

IV. CHRONIC AFFECTIONS OF THE SPINAL CORD.

I. Spastic Paraplegia.

Definition.—Loss of power with spasm of the muscles of the lower extremities.

While clinically spastic paraplegia, or, as it is sometimes called, *tabes dorsalis spasmodique*, is a well-defined, readily recognizable affection, etiologically and anatomically it presents marked differences, and various groups must be separated, all of which present, however, the combination of spasm with loss of power. As the pyramidal tracts are involved, the term lateral sclerosis is sometimes used as the equivalent of spastic paraplegia. I shall consider the following forms:

look up

In the syphilitic form described by Erb. the bladder is involved. There are slight disturbances of sensation. The lesion is probably a transverse myelitis in the dorsal region or more probably a meningo-myelitis. Erb says that in some cases there is a tendency to improvement under specific treatment. (Med Chron. April 84

(1) **Secondary Spastic Paralysis.**—After any tranverse lesion of the cord, whether the result of slow compression (as in caries), chronic myelitis, the pressure of tumor, chronic meningo-myelitis, or multiple sclerosis, degeneration takes place in the pyramidal tracts below the point of disease. The legs soon become stiff and rigid, and the reflexes increase. It happens occasionally, as Bastian has shown, that in compression paraplegia the limbs may be flaccid without increase in the reflexes—*paraplegie flasque* of the French. The condition of the patient in these secondary forms varies very much. In chronic myelitis or in multiple sclerosis he may be able to walk about, but with a characteristic spastic gait. In the compression myelitis, in fracture, or in caries, there may be complete loss of power with rigidity.

(2) **Primary Spastic Paraplegia.**—This is believed to depend upon a primary sclerosis of the lateral or pyramidal tracts. Clinically it is common to meet with cases in adults, particularly in syphilitic subjects, who have pains in the back, perhaps a girdle sensation, and a gradually developing, progressive spastic paraplegia. It may be impossible from the history or the physical examination to determine whether the condition is secondary to a transverse myelitis or a meningo-myelitis, or whether the lesion is a primary degeneration of the pyramidal tracts. The question is still debated whether a primary lesion of the lateral tracts ever takes place, or whether, in such instances, there is not always some lesion of the motor cells in the anterior horns. Cases may persist for years without any atrophy. In other instances there are signs of involvement of the posterior columns as well, forming the condition of ataxic paraplegia, which will be considered separately. So far as I know, the only case which is claimed to demonstrate the existence of a primary lateral sclerosis is that of Dreschfeld's, which occurred in 1881.

The *symptoms* of spastic paraplegia are very distinctive. The patient complains of feeling tired, of stiffness in the legs, and perhaps of pains of a dull aching character in the back or in the calves. There may be no definite loss of power, even when the spastic condition is well established. In other instances there is definite weakness. The stiffness is felt most in the morning. In a well-developed case the gait is most characteristic. The legs are moved stiffly and with hesitation, the toes drag and catch against the ground, and, in extreme cases, when the ball of the foot rests upon the ground a distinct clonus develops. The legs are kept close together, the knees touch, and in certain cases the adductor spasm may cause cross-legged progression. On examination, the legs may at first appear tolerably supple, perhaps flexed and extended readily. In other cases the rigidity is marked, particularly when the limbs are extended. The spasm of the adductors of the thigh may be so extreme that the legs are separated with the greatest difficulty. In cases of this extreme rigidity the patient usually loses the power of walking. The nutrition is well maintained, the muscles may be hypertrophied. The reflexes are greatly

increased. The slightest touch upon the patellar tendon produces an active knee-jerk. The rectus clonus and the ankle clonus are easily obtained. In some instances the slightest touch may throw the legs into violent clonic spasm, the condition to which Brown-Séquard gave the name of spinal epilepsy. The superficial reflexes are also increased. The arms may be unaffected for years, but as a late manifestation rigidity may develop.

The *diagnosis* is readily made, but it is often very difficult to determine accurately the nature of the underlying pathological condition. A history of syphilis is present in many of the cases. The course of the disease is progressively downward. Years may elapse before the patient is bed-ridden. Involvement of the sphincters, as a rule, is late; occasionally, however, it is early. The sensory symptoms rarely progress and the patients may retain the general nutrition and enjoy excellent health. Ocular symptoms are rare.

(3) **The Spastic Paraplegia of Infants** (*Paraplegia Cerebralis Spastica* —Heine).—This is usually a birth palsy, often the result of difficult labor. In twenty-three of the twenty-four of Little's cases, there was either difficult labor or premature delivery. Several children may be affected in a family. Gee reports two cases in one family, Schultze three, and with Latimer I saw a brother and a sister with the disease. In this connection it is interesting to note that Bernhardt has recently described a family form of spastic paraplegia, in which four brothers were affected, the disease developing in each about the thirtieth year. The stiffness of the legs may not be noticed for some months after birth, but usually on dressing the child the mother notices the rigidity. When attempts are made to walk the stiffness and awkwardness then become apparent. On standing, the attitude is very characteristic. There is talipes equinus, varying from the slightest raising of the heel to a condition in which the child stands on tiptoe. In older children, as they walk, the toe-cap of the shoe is usually much worn. The strong adductor action may produce typical cross-legged progression, in which each foot is dragged over and planted in front, or even on the other side of its fellow. In attempting to flex the legs there is a marked resistance, which gradually yields—the lead-pipe contraction, as Weir Mitchell calls it. The reflexes are increased, though in some children it is not an easy matter to obtain them. The ankle clonus, as a rule, is not obtainable. Sensation is unimpaired, and the bladder and rectum are not involved.

The symptoms of this affection in children are almost identical with the spastic paraplegia of adults. The arms may be involved—spastic diplegia. The disease is probably of cortical origin. There are frequently symptoms indicating cerebral defects, such as idiocy, imbecility, and nystagmus. Some of the cases depend, no doubt, upon bilateral meningeal hæmorrhage occurring during delivery. Others are probably due to arrest of development of the pyramidal tracts. This condition in children must

not be confounded with tetany or with the pseudo-paralytic rigidity so often associated with rickets.

(5) **Ataxic Paraplegia.**—This name is applied by Gowers to a disease characterized clinically by a combination of ataxia and spastic paraplegia, and anatomically by involvement of the posterior and lateral columns.

The disease is most common in middle-aged males. Exposure to cold and traumatism have been occasional antecedents. In striking contrast to ordinary tabes a history of syphilis is rarely to be obtained.

The anatomical features are a sclerosis of the posterior columns, which is not more marked in the lumbar region and not specially localized in the root zone of the postero-external columns. The involvement of the lateral columns is diffuse, not always limited to the pyramidal tracts, and there may be an annular sclerosis.

The *symptoms* are well defined. The patient complains of a tired feeling in the legs, not often of actual pain. The sensory symptoms of true tabes are absent. An unsteadiness in the gait gradually develops with progressive weakness. The reflexes are increased from the outset, and there may be well-developed ankle clonus. Rigidity of the legs slowly comes on, but is rarely so marked as in the uncomplicated cases of lateral sclerosis. From the start, incoördination is a well-characterized feature, and the difficulty of walking in the dark or swaying when the eyes are closed may, as in true tabes, be the first symptom to attract attention. In walking the patient uses a stick, keeps the eyes fixed on the ground, the legs far apart, but the stamping gait, with elevation and sudden descent of the feet, is not often seen. The incoördination may extend to the arms. Sensory symptoms are rare, but Gowers calls attention to a dull, aching pain in the sacral region. The sphincters usually become involved. Eye symptoms are rare. Late in the disease mental symptoms may develop, similar to those of general paresis.

In well-marked cases the *diagnosis* is easy. The combination of marked incoördination with retention of the reflexes and more or less spasm are characteristic features. The absence of ocular and sensory symptoms is an important point.

(6) **Hysterical Spastic Paraplegia.**—There is no spinal-cord disease which may be so accurately mimicked by hysterical patients as spastic paraplegia. There is wasting in the hysterical paraplegia, the sensory symptoms are not marked, the loss of power is not complete, and there is not that extensor spasm so characteristic of organic disease. The hysterical contracture will be considered later.

The reflexes are, as a rule, increased. The knee-jerk is present, and there may be well-developed ankle clonus. Gowers calls attention to the fact that it is usually a spurious clonus, "due to a half-voluntary contraction in the calf muscles." A true clonus does occur, however, and there may be the greatest difficulty in determining whether or not the case is one of hysterical paraplegia.

(§) **Primary Combined Sclerosis** (Putnam).—In addition to the ataxic paraplegia just mentioned, here may be considered certain cases which are characterized anatomically by a relatively chronic sclerosis of the posterior columns, of the lateral columns, chiefly the pyramidal tract, and also of the cerebellar tract. With these are usually associated more acute changes in adjoining areas, either diffuse or systemic, some grade of degeneration in the gray matter, and involvement of the nerve roots. This form has been studied by J. J. Putnam and Dana. The cases are usually in women—seven out of nineteen collected by Dana; the ages, from forty-five to sixty-four. The disease runs a rather rapid course. Neuropathic inheritance is present in some instances. Putnam thinks that possibly both lead and arsenic play a part in the etiology.

The *symptoms* are both sensory and motor. The onset is usually with numbness in the extremities, progressive loss of strength, and emaciation. Paraplegia gradually develops, before which there have been, as a rule, spastic symptoms with exaggerated knee-jerk. The arms are affected less than the legs. Mental symptoms similar to dementia paralytica may develop toward the close.

The *diagnosis* of this mixed sclerosis rests upon the combination of sensory and motor symptoms with the presence of exaggerated reflexes. As stated, the sensory features consist chiefly of paræsthesia, and there may be difficulty in distinguishing the condition from multiple neuritis. The frequency of the disease in more or less enfeebled or anæmic women past middle life is also an important feature.

Treatment of Spastic Paraplegia.—In the majority of cases spastic paraplegia is incurable. The cases which result from transitory compression, as in caries, may get well; but in the other forms the disease is uniformly progressive, and remedies have little or no control. When syphilis is suspected a thorough course of mercury and iodide of potassium should be given. Scrupulous attention should be paid to the bladder symptoms, and the same measures may be used as will be advised in locomotor ataxia. In the infantile form of paraplegia much may be done by the orthopædic surgeon to overcome rigidity and contracture. In several instances I have known persistent friction with forcible flexion and extension and the application of proper apparatus enable a patient to get about comfortably.

II. Locomotor Ataxia

(*Tabes Dorsalis; Posterior Spinal Sclerosis*).

Definition.—An affection of the nervous system characterized clinically by incoördination, with sensory and trophic disturbances and involvement of the special senses, particularly the eyes. Anatomically there are found sclerosis of the posterior columns of the cord, foci of degeneration in the basal ganglia, and sometimes chronic degenerative changes in the cortex cerebri.

Etiology.—It is a wide-spread disease, more frequent in cities than in the country. The relative proportion may be judged from the fact that of 1,816 cases in my neurological dispensary in two years there were 25 cases of locomotor ataxia. Males are attacked more frequently than females, the proportion being at least ten to one. Mitchell has called attention to the fact that it is a rare disease in the negro. Of 25 cases at my clinic, 3 were in negroes. It is a disease of adult life, a majority of the cases occurring between the thirtieth and fortieth years. Occasionally cases are seen in young men. The form of ataxia which occurs in children is a different disease. Of special causes syphilis is the most important. According to the figures of Erb, Fournier, and Gowers, in from fifty to seventy-five per cent of all cases there is a history of this disease. Erb's recent figures are most striking; of 300 cases of tabes in private practice 89 per cent had had syphilis.

Excessive fatigue, overexertion, exposure to cold and wet, and sexual excesses are all assigned as causes. There are instances in which the disease has closely followed severe exposure. James Stewart has noted that the Ottawa lumbermen, who live a very hard life in the camps during the winter months, are frequently the subjects of locomotor ataxia. Trauma has been noted in a few cases. Alcoholic excess does not seem to predispose to the disease. Among patients in the better classes of life I do not remember one in which there had been a previous history of prolonged drunkenness.

Morbid Anatomy.—When a patient has died in the advanced stage of the disease the following are the most important changes:

(*a*) The peripheral nerves may show signs of degeneration. Neuritis may indeed be present even when there have been no special symptoms indicating it. In other instances there is not only neuritis, but muscular atrophy.

(*b*) The posterior roots of the spinal cord are small, gray, and atrophic.

(*c*) The meninges of the posterior and lateral columns are thickened, more firmly adherent than normally, and the blood-vessels usually show signs of arterio-sclerosis.

(*d*) The changes in the spinal cord are as follows: (1) In advanced cases the posterior columns are uniformly sclerotic and the dorsal and lumbar regions are most extensively involved. In long-standing cases there is generally an increase of connective tissue throughout the cord and there may be degeneration (2) of the ascending antero-lateral tract; (3) of the direct cerebellar tract; (4) of the pyramidal tract.

(*e*) In early cases the course of the anatomical changes may be traced. The steps in the process are as follows: The posterior root-zone of Charcot is first involved, often with the fibres of the posterior root, so that it has been thought to begin perhaps as a neuritis of these roots within the vertebral canal. The narrow strip which lies between the pyramidal tract

and the posterior cornu, known as Lissauer's tract, is early involved, together with the nerve-cells of the adjacent Clarke's vesicular column. In what is known as the pre-ataxic stage these may be the only alterations. Subsequently the sclerosis extends widely in the postero-external, and subsequently in the postero-median columns.

(*f*) The cerebral changes—of less consequence than the spinal—may consist of (1) sclerosis in the restiform bodies, in the inferior peduncles of the cerebellum, and of certain of the cranial nerves, particularly the third, the optic, and the auditory; (2) cortical changes, consisting in some cases of a diffuse meningo-encephalitis.

Symptoms.—These are best considered under the three stages of pre-ataxic, ataxic, and paralytic.

Pre-ataxic Stage.—The following are the most characteristic features ol this period:

Pains, usually of a sharp stabbing character; hence the term, lightning pains. They last for only a second or two and are most common in the legs. They may be associated with a hot, burning feeling. Occasionally herpes may develop at the site of the pain. They may occur at irregular intervals, and are more prone to follow excesses or to come on when the health is impaired.

Ocular Symptoms.—(*a*) Ptosis, which may be single or double and is by no means uncommon either alone or (*b*) in association with external strabismus. The first complaint may be of double vision. Occasionally there may be paralysis of all the external muscles of the eye, producing ophthalmoplegia externa. (*c*) Argyll-Robertson pupil, in which, as already mentioned, there is loss of the iris reflex to light, but contraction during accommodation. The pupils are usually small—spinal myosis. (*d*) Optic atrophy. This is often an early, or even the first symptom. The loss of vision progresses, and in a large majority of cases leads to total blindness.

Loss of the Knee-jerk.—This is one of the earliest symptoms, and may occur years before there is ataxia. Taken alone it is of no moment, as there are individuals in whom the knee-jerk is absent; but in connection with the lightning pains and the ocular symptoms, it is of special importance. These are the most common symptoms of the pre-ataxic stage, and may persist for years without the development of incoördination. The patient may look well and feel well, and be troubled only by occasional attacks of lightning pains; or there is persistent ptosis, external strabismus develops, or, what is more serious, a progressive atrophy of the optic nerve. There is often a gradual loss of sexual power.

The disease may never progress beyond this stage, and when optic atrophy develops early and leads to blindness, the ataxia rarely, if ever, supervenes. There is a sort of antagonism between the ocular symptoms and the progress of the ataxia. Charcot lays considerable stress upon this, and Déjérine assured me that of the enormous tabetic material at the

The patient may be quite unable to stand with the legs.

He does not start off promptly at the word of command,
He goes descends slowly & with difficulty.

Bicêtre in not a single instance in which optic atrophy had come on early and progressed to blindness was the patient ataxic, although there were cases which had had the lightning pains and lesions of the optic nerves for twenty-five years.

Ataxic Stage.—*Motor Symptoms.*—The ataxia develops gradually. One of the first indications to the patient is inability to get about readily in the dark or to maintain his equilibrium when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position. This is known as Romberg symptom. On turning quickly he is apt to fall. Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick, the eyes are directed to the ground, the body is thrown forward, and the legs are wide apart. In walking, the leg is thrown out violently, the foot is raised too high and is brought down in a stamping manner with the heel first, or the whole sole comes in contact with the ground. Ultimately the patient may be unable to walk without the assistance of two canes. This gait is very characteristic, and unlike that seen in any other disease. The incoördination is not only in walking, but in the performance of other movements. If the patient is asked, when in the recumbent posture, to touch the knee with one foot, the irregularity in the movement is very evident. Incoördination of the arms is less common, but usually develops in some grade. It may in rare instances exist before the incoördination of the legs. In the large number of ataxics which frequented the Infirmary for Nervous Diseases at Philadelphia, there was only one, so far as I remember—at Weir Mitchell's clinic—in which the arms were first affected. It may be tested by asking the patient to close his eyes and to touch the tip of the nose or the tip of the ear with the finger, or with the arms thrust out to bring the tips of the fingers together. The incoördination may early be noticed by a difficulty which the patient experiences in buttoning his collar or in performing one of the ordinary routine acts of dressing.

One of the most striking features of the disease is that with marked incoördination there is no loss of muscular power. The grip of the hands may be strong and firm, the power of the legs, tested by trying to flex them, may be unimpaired, and their nutrition, except toward the close, may be unaffected.

Sensory Symptoms.—The lightning pains may persist. They vary greatly in different cases. Some patients are rendered miserable by the frequent occurrence of the attacks; others escape altogether. In addition, common symptoms are tingling, pins and needles, particularly in the feet, and areas of hyperæsthesia or of anæsthesia. The patient may complain of a change in the sensation in the soles of the feet, as if cotton was interposed between the floor and the skin. Sensory disturbances occur less frequently in the hands. Retardation of tactile sensation is common, and a pin-prick on the foot, instead of being instantaneously felt, is not per-

ceived for a second or two or may be delayed for as much as ten seconds. The pain felt may persist. A curious phenomenon is the loss of the power of localizing the pain. For instance, if the patient is pricked on one limb he may say that he feels it on the other (allocheiria), or a pin-prick on the foot may be felt in both feet. The muscular sense becomes much impaired and the patient no longer recognizes the position in which his limbs are placed. This may be present in the pre-ataxic stage.

Reflexes.—As mentioned, the loss of the knee-jerk is one of the earliest symptoms of the disease. Occasionally a case is found in which it is retained. The skin reflexes may at first be increased, but later are usually involved with the deep reflexes.

Special Senses.—The eye symptoms noted above may be present, but, as mentioned, ataxia is rare with atrophy of the optic nerve.

Deafness may develop, due to lesion of the auditory nerve. There may also be attacks of vertigo. Olfactory symptoms are rare.

Visceral Symptoms.—Among the most remarkable sensory disturbances are the tabetic crises, severe paroxysms of pain referred to various viscera; thus laryngeal, gastric, nephralgic, rectal, urethral, and clitoral crises have been described. The most common are the gastric and laryngeal. In the former there are intense pains in the stomach, vomiting, and a secretion of hyperacid gastric juice. The attack may last for several days or even longer. There may be severe pain without any vomiting. The attacks are of variable intensity and usually require morphia. Paroxysms of rectal pain and tenesmus are described. They have not been common in my experience. Laryngeal crises also are rare. There may be true spasm with dyspnœa and noisy inspiration. In one instance at least the patient has died in the attack.

The sphincters are frequently involved. Early in the disease there may be a retardation or hesitancy in making water. Later there is retention, and cystitis may occur. Unless great care is taken the inflammation may extend to the kidneys. Constipation is extremely common. Late in the disease the sphincter ani is weakened. The sexual power is usually lost in the ataxic stage.

Trophic Changes.—Skin rashes may develop in the course of the lightning pains, such as herpes, œdema, or local sweating. Alteration in the nails may occur. A perforating ulcer may develop on the foot, usually beneath the great toe. Onychia may prove very troublesome.

The arthropathies or joint lesions affect chiefly the knees. They are unquestionably associated with the disease itself, and not necessarily a result of trauma. The condition, known as Charcot's joint, is anatomically similar to that of chronic arthritis deformans. The effusion may be rapid and there may be great disintegration and destruction of the cartilages and bones, leading to dislocation and deformity. Pus was present in a well-marked Charcot's joint in a patient of C. K. Mills at the Philadelphia Hospital. Spontaneous fractures may occur. Among other trophic

disturbances may be mentioned atrophy of the muscles, which is usually a late manifestation, but may be localized and associated with neuritis. In any very large collection of cases many instances of atrophy are found, due either to involvement of the anterior horns or to peripheral neuritis.

Cerebral Symptoms.—Hemiplegia may develop at any stage of the disease, more commonly when it is well advanced. It may be due to hæmorrhagic softening in consequence of disease of the vessels or to progressive cortical changes. Hemianæsthesia is sometimes present. Very rarely the hemiplegia is due to coarse syphilitic disease.

Dementia paralytica frequently exists with tabes, and it may be extremely difficult to determine which has been the primary affection. In a majority of the cases the locomotor ataxia has preceded the symptoms of general paresis. In other instances melancholia, dementia, or paranoia develop.

(*c*) **Paralytic Stage.**—After persisting for an indefinite number of years the patient gradually loses the power of walking and becomes bedridden or paralyzed. In this condition he is very likely to be carried off by some intercurrent affection, such as pyelo-nephritis, pneumonia, or tuberculosis.

The Course of the Disease.—A patient may remain in the pre-ataxic stage for an indefinite period, and the loss of knee-jerk and the gray atrophy of the optic nerves may be the sole indications of the true nature of the disease. In such cases incoördination rarely develops. In a majority of cases the progress is slow, and after six or eight years, sometimes less, the ataxia is well developed. The symptoms may vary a good deal; thus the pains, which may have been excessive at first, often lessen. The disease may remain stationary for years; then exacerbations occur and it makes rapid progress. Occasionally the disease seems to be arrested. There are instances of what may be called acute ataxia, in which, within a year or even less, the incoördination is marked, and the paralytic stage may develop within a few months. The disease itself rarely causes death, and after becoming bedridden the patient may live for fifteen or twenty years.

Diagnosis.—In the pre-ataxic stage the combination of lightning pains and the absence of knee-jerk is distinctive. The association of progressive atrophy of the optic nerves with loss of knee-jerk is also characteristic. The early ocular palsies are of the greatest importance. A squint, ptosis, or the Argyll-Robertson pupil may be the first symptom, and may exist with the loss only of the knee-jerk. Loss of the knee-jerk alone, however, does occasionally occur in healthy individuals.

The diseases most likely to be confounded with locomotor ataxia are: (1) *Peripheral Neuritis.*—The pseudo-tabetic gait of arsenical, alcoholic, or diabetic paralysis is quite unlike that of locomotor ataxia. In these forms there is a paralysis of the feet and the leg is lifted high in order that the toes may clear the floor. The use of the word tabes in this con-

nection should no longer be continued. If in any doubt, the absence of the lightning pains and eye symptoms and the history will suffice in the majority of cases to make the diagnosis clear. In diphtheritic paralysis the early loss of knee-jerk and the associated eye symptoms may suggest tabes, but the history, the existence of paralysis of the throat, and the absence of pains render a diagnosis easy.

(2) *Ataxic Paraplegia.*—Marked incoördination with spastic paralysis is characteristic of the condition which Gowers has termed ataxic paraplegia. In a majority of the cases this affection is distinguished also by the absence of pains and of eye symptoms.

(3) *Cerebellar Disease.*—The cerebellar incoördination has only a superficial resemblance to that of locomotor ataxia; the knee-jerk is present, there are no lightning pains, no sensory disturbances; while, on the other hand, there are headache, optic neuritis, and vomiting.

(4) Some *acute affections* involving the posterior columns of the cord may be followed by incoördination and resemble tabes very closely. In a case recently under my care, the gait was characteristic and Romberg's symptom was present. The knee-jerk, however, was retained and there were no ocular symptoms. The condition had developed within three or four months, and there was a well-marked history of syphilis. Under large doses of iodide of potassium the ataxia and other symptoms completely disappeared.

(5) *General Paresis.*—In some cases this offers a serious difficulty. In the first place, in general paresis, tabetic symptoms often develop; on the other hand, there are cases of locomotor ataxia in which, toward the end, there are symptoms of general paresis. Cases of unusually acute ataxia with mental symptoms belong, as a rule, to the former disease. The question will be considered under general paresis.

(6) Visceral crises and neuralgic symptoms may lead to error, and in middle-aged men with severe, recurring attacks of gastralgia it is always well to bear in mind the possibility of tabes, and to make a careful examination of the eyes and of the knee-jerk.

Prognosis.—Complete recovery cannot be expected, but arrest of the progress is not uncommon and a marked amelioration of the symptoms is frequent. Optic-nerve atrophy, one of the most serious events in the disease, has this hopeful aspect—that incoördination rarely follows and the progress may be arrested. The optic atrophy itself is occasionally checked. On the whole, the prognosis in tabes is bad. The experience of such men as Weir Mitchell, Charcot, and Gowers is distinctly opposed to the belief that locomotor ataxia is ever completely cured.* No such instance has come under my personal observation.

Treatment.—To arrest the progress and to relieve, if possible, the symptoms are the objects which the practitioner should have in view. A

*For a study of the reputed cures, see L. C. Gray, N. Y. Medical Journal, Nov., 1889.

quiet, well-regulated method of life is essential. It is not well, as a rule, for a patient to give up his occupation so long as he is able to keep about and perform ordinary work. I know tabetics who have for years conducted large businesses, and there have been several notable instances in our profession of men who have risen to distinction in spite of the existence of this disease. Excesses of all sorts, more particularly *in baccho et venere*, should be carefully avoided. A man in the pre-ataxic stage should not marry.

Care should be taken in the diet, particularly if gastric crises have occurred. To secure arrest of the disease many remedies have been employed. Although syphilis plays such an important *rôle* in the etiology, it is universally acknowledged that neither mercury nor the iodide of potassium have as a rule the slightest influence over the tabetic lesions. To this there is but one exception—when the syphilis is comparatively recent; when the symptoms develop within two years of the primary infection, there is then a possibility of arrest by mercury and iodide of potassium. However, they do not always relieve. In two cases of very rapidly progressing tabes following syphilis this medication was of no avail. Not only is an anti-~~mercurial~~ treatment of no benefit in the majority of cases of locomotor ataxia, but my experience tallies with that of Gowers in that it may even hasten the progress of the disease. Of remedies which may be tried and are believed by some writers to retard the progress, the following are recommended: Arsenic in full doses, nitrate of silver in quarter-grain doses, Calabar bean, ergot, and the preparations of gold.

The treatment by suspension introduced a few years ago has already been practically abandoned. Good effects certainly have followed in a few cases, but it was unreasonable from the outset, either on therapeutic or scientific grounds, to hope that by such a measure permanent changes could be induced in the pathological condition. The benefits were due in great part to suggestion and to psychical effects. In any case it must be used with caution.

For the pains, complete rest in bed, as advised by Weir Mitchell, and counter-irritation to the spine (either blisters or the thermo-cautery) may be employed. The severe spells which come on particularly after excesses of any kind are often promptly relieved by a hot bath or by a Turkish bath. A prolonged course of nitrate of silver seems in some cases to allay the pains and lessen the liability to the attacks. I have never seen ill effects from its use in the spinal scleroses. Antipyrin and antifebrin may be employed, and occasionally do good, but their analgesic powers in this disease have been greatly overrated. Cannabis indica is sometimes useful. In the severe paroxysms of pain hypodermics of morphia or of cocaine must be used. The use of morphia should be postponed as long as possible. Electricity is of very little benefit. For the severe attacks of gastralgia, morphia is also required. The laryngeal crises are rarely dangerous. An application of cocaine may be made during the spasm, or a few whiffs of chloroform may be given, or nitrite of amyl. In all cases of tabes

with increased arterial tension the prolonged use of nitroglycerin, given in increasing doses until the physiological effect is produced, is of great service in allaying the neuralgic pains and diminishing the frequency of the crises. Its use must be guarded when there is aortic insufficiency. The special indication is increased tension. The bladder symptoms demand constant care. When the organ cannot be perfectly emptied the catheter should be used, and the patient may be taught its use and how to keep it thoroughly sterilized.

III. Hereditary Ataxia (*Friedreich's Ataxia*).

In 1861 Friedreich reported six cases of a form of hereditary ataxia, and the affection has usually gone by his name. Unfortunately, *paramyoclonus multiplex* is also called Friedreich's disease; so it is best, if his name is used in connection with this affection, to term it Friedreich's ataxia. It is a very different disease in many respects from ordinary tabes. It may or may not be hereditary. It is really a family disease, several brothers and sisters being, as a rule, affected. The 143 cases analyzed by Griffith occurred in 71 unrelated families. In his series inheritance of the disease itself occurred in only 33 cases. Various influences in the parents have been noted; alcoholism in only 7 cases. Syphilis has rarely been present. Of the 143 cases, 86 were males and 57 females. The disease sets in early in life, and in Griffith's series 15 occurred before the age of two years, 39 before the sixth year, 45 between the sixth and tenth years, 20 between the eleventh and fifteenth years, 18 between the sixteenth and twentieth years, and 5 between the twentieth and twenty-fifth years.

The *morbid anatomy* shows an extensive sclerosis of the posterior and lateral columns of the spinal cord. The periphery, and the cerebellar tracts are usually involved. The recent observations of Déjérine and Letulle are of special interest, since they seem to indicate that the change in this disease is a neurogliar (ectodermal) sclerosis, differing entirely from the ordinary spinal sclerosis. According to this view, Friedreich's disease is a gliosis of the posterior columns due to developmental errors.

Symptoms.—The ataxia is unlike the ordinary form. The incoordination begins in the legs, but the gait is peculiar. It is swaying, irregular, and more like that of a drunken man. There is not the characteristic stamping gait of the true tabes. Romberg's symptom may or may not be present. The ataxia of the arms occurs early and is very marked; the movements are almost choreiform, irregular, and somewhat swaying. In making any voluntary movement the action is overdone, the prehension is claw-like, and the fingers may be spread or overextended just before grasping an object. The hand frequently moves about an object for a moment and then suddenly pounces upon it. There are irregular, swaying movements, some of which are choreiform, of the head

There is a form of hereditary ataxia described by Marie as hereditary cerebellar heredo-ataxy which starts later in life, after the age of twenty, with disability in the legs but the gait is less ataxic than 'groggy'. The knee jerks are exaggerated and a spastic gait of the legs ultimately develops. The cerebellum has been found atrophied in two cases. (La Semaine Med. Oct 1893)

There is no scoliosis nor does club foot develop. Some known cases, twenty five in one family, and the Illoff's, thirteen, appear to belong to this type

It is much commoner in persons under thirty years of age, and of 190 cases 133 were in men 57 in women (Schlesinger) A large majority of the cases begin before the 30th year. The disease has been met with in three members of the same family (Rev. Neurology 1895.)

and shoulders. There is present in many cases what is known as static ataxia, that is to say, ataxia of quiet action—irregular, slow movements of the fingers or the hands while at rest.

Sensory symptoms are not usually present. The reflexes may be lost. In Griffith's table they were abolished in 91 cases.

Nystagmus is a characteristic symptom. Atrophy of the optic nerve rarely occurs. A striking feature is early deformity of the feet. There is talipes equinus, and the patient walks on the outer edge of the feet. The big toe is flexed dorsally on the first phalanx. Lateral curvature of the spine is very common.

Trophic lesions are rare. As the disease advances paralysis comes on and may ultimately be complete. Some of the patients never walk.

Disturbance of speech is common. It is usually slow and scanning; the expression is often dull; the mental power is, as a rule, maintained, but late in the disease becomes impaired.

The *diagnosis* of the disease is not difficult when several members of a family are affected. The onset in childhood, the curious form of incoördination, the early talipes equinus, the position of the great toe, the scoliosis, the nystagmus, and scanning speech make up an unmistakable picture. The disease is often confounded with chorea, with the ordinary form of which it has nothing in common. With hereditary chorea it has certain similarities, but usually this disease does not set in until after the thirtieth year.

The disease lasts for many years and is incurable. Care should be taken to prevent contractures.

IV. Syringo-myelia.

Definition.—A gliomatous new formation about the central canal of the spinal cord, with cavity formation.

The disease has attracted a good deal of attention within the past few years, and has a definite clinical interest since cases can now be diagnosed.

Etiology and Morbid Anatomy.—Syringo-myelia must be distinguished from dilatation of the central canal—hydromyelus—slight grades of which are not very uncommon either as a congenital condition or as a result of the pressure of tumors. The cavity of syringo-myelia has a variable extent in the cord, sometimes existing in the entire length, but in many cases involving only the cervical and dorsal regions or a more limited area. It is usually in the posterior portion of the cord and extends into one posterior cornu. The transverse section may be oval or circular or narrow and fissure-like. It varies at different levels. The condition is now regarded as a *gliosis*, a development of embryonal neurogliar tissue in which hæmorrhage or degeneration takes place with the formation of cavities.

Symptoms.—The disease, which is of slow development, makes its appearance, as a rule, about adolescence, and may persist for fifteen or twenty years. There are irregular pains, chiefly in the cervical region; muscular atrophy develops, which may be confined to the arms, or sometimes extends to the legs. The reflexes are increased and a spastic condition develops in the legs. Ultimately the clinical picture may be that of an amyotrophic lateral sclerosis. The tactile sensation is usually intact and the muscular sense is retained, but painful and thermic sensations are not recognized, or there may be in rare instances complete anæsthesia of the skin and of the mucous membranes (Déjérine). This combination of loss of painful and thermic sensations with paralysis of an amyotrophic type is regarded as pathognomonic of the disease. The special senses are usually intact and the sphincters uninvolved. Trophic troubles are not uncommon. Owing to the loss of the painful and heat sensations, the patients are apt to injure themselves. A man aged seventy, whom I saw with Déjérine at the Bicêtre, had had the symptoms for over twenty-five years. Loss of sensation had preceded the atrophy, and the terminal phalanx of the middle finger was charred, as he experienced no sensation whatever when the hot end of the cigarette neared his finger. Scoliosis also may be present in these cases. The loss of painful and thermic impressions is due to the fact that these pass to the brain in the peri-ependymal gray matter, particularly that portion in the posterior roots, which is almost constantly involved in syringo-myelia. The tactile sensation is retained because the postero-external column is uninvolved.

In typical cases the *diagnosis* is easy. The combination of an amyotrophic paralysis, the picture of progressive muscular atrophy of the Aran-Duchenne type, with retention of tactile and loss of thermic and painful sensation, is probably pathognomonic of the disease. Of affections with which it may be confounded, anæsthetic leprosy is the most important, since the anæsthesia and the wasting may closely simulate it; but, as a rule, in leprosy trophic changes are more or less marked. There is often loss of phalanges and there is no characteristic dissociation of sensory impressions.

There is a remarkable affection confined to a district of Brittany and known as Morvan's disease, after the physician who described it. The disease is chronic and characterized by neuralgic pains, cutaneous anæsthesia, and painless and destructive whitlows. In Gombault's autopsy neuritis was found, but it could not be decided, owing to the state of the cord when examined, whether cavities existed or not. Joffroy reports a case in which syringo-myelia was present and claims the affections are identical. The curious distribution of the disease and the fact that at least 20 cases have occurred in a population of 5,000, suggest that it is possibly a peripheral neuritis of infectious origin. Church, of Chicago, has reported case in which, with features believed to be characteristic of syringo-myelia, the patient had the painless and destructive whitlows which form so special a feature in Morvan's disease.

The clinical features are extremely complex. In the classical form

Schlesinger, in his recent monograph (1895) recognizes the following types; (1), with the classical features above described, which may begin in the cervical or lumbar regions; (2), a motor type, with the picture of an amyotrophic or a spastic paralysis; the sensation may be undisturbed for years; (3), with predominant sensory features, simulating hysterical hemiplegia, or with the general anæsthesia of pain & temperature; (4), with pronounced trophic disturbances. To this type belong the cases described as Morvan's disease, an affection characterized by neuralgic pains, cutaneous anæsthesia and painless destructive whitlows. (5), the tabetic type, either a combination of tabetic symptoms with those of tabes and syringomyelia in the upper extremities or a pure tabetic symptom complex due to invasion of the fibres of the posterior columns (Oppenheim). Arthropathies occur in about 10 per cent of the cases 130

Some of the cases of Morvan's disease have been those of leprosy.

a

Fatal haemorrhage may follow erosion
The vertebral artery ~~may be obliterated~~

A cervico-brachial neuralgia may be an
early symptom

In regions where skin anesthetic to
tactile & painful impressions

V. Compression of the Spinal Cord (*Compression Myelitis*).

Definition.—Interruption of the functions of the cord by slow compression.

Etiology.—Caries of the spine, new growths, aneurism, and parasites are the important causes of slow compression. Caries, or Pott's disease, as it is usually called, after the surgeon who first described it, is in the great majority of instances a tuberculous affection. In a few cases it is due to syphilis and occasionally to extension of disease from the pharynx. It is most common in early life, but may occur after middle age. It follows trauma in a few cases. Compression occasionally results from aneurism of the thoracic aorta or the abdominal aorta, in the neighborhood of the cœliac axis.

Malignant growths frequently cause a compression paraplegia. A retroperitoneal sarcoma or the lymphadenomatous growths of Hodgkin's disease may invade the vertebræ. More commonly, however, the involvement is secondary to scirrhus of the breast.

Of parasites, the echinococcus and the cysticercus occasionally occur in the spinal canal.

Symptoms.—These may be considered as they affect the bones, the nerves, and the cord.

(1) **Vertebral.**—In malignant disease and in aneurism erosion of the bodies may take place without producing any deformity of the spine. In caries, on the other hand, it is the rule to find more or less deformity, amounting often to angular curvature. The compression is largely due to the thickening of the dura and the presence of caseous and inflammatory products between this membrane and the bone. The compression is rarely produced directly by the bone. Pain is a constant and, in the case of aneurism and tumor, agonizing feature. In caries, the spinal processes of the affected vertebræ are tender on pressure, and pain follows jarring movements or twisting of the spine. There may be extensive tuberculous disease without much deformity, particularly in the cervical region.

(2) **Nerve-root Symptoms.**—These result from compression of the nerve-roots as they pass out between the vertebræ. It is remarkable how frequently, even in extensive caries, they escape and the patient does not complain of radiating pains in the distribution of the nerves from the affected segment. Pains are more common in cancer of the spine secondary to that of the breast, and in such cases may be agonizing. There may be acutely painful areas ~~of hyperæsthesia of the skin or anæsthesia~~—the *anæsthesia dolorosa*. Trophic disturbances may occur, particularly herpes. In the cervical or lumbar regions pressure on the anterior roots may give rise to wasting of the muscles supplied by the affected nerves.

(3) **Cord Symptoms.** (*a*) *Cervical Region.*—Not infrequently the caries is high up between the axis and the atlas or between the latter and the occipital bone. In such instances a retropharyngeal abscess may be

present, giving rise to difficulty in swallowing. There may be spasm of the cervical muscles, the head may be fixed, and movements may either be impossible or cause great pain. In a case of this kind in the Montreal General Hospital movement was liable to be followed by transient, instantaneous paralysis of all four extremities, owing to compression of the cord. In one of these attacks the patient died.

In the lower cervical region there may be signs of interference with the cilio-spinal centre and dilatation of the pupils. Occasionally there is flushing of the face and ear of one side or unilateral sweating. Deformity is not so common, but healing may take place with the production of a callus of enormous breadth, and complete rigidity of the neck. The nerves of the upper extremities may be involved, and shooting pains may occur in the arm.

(*b*) *Dorsal Region.*—The deformity is here more marked and pressure symptoms are more common. The time of onset of the paralysis varies very much. It may be an early symptom, even before the curvature is manifest. More commonly it is late, occurring many months after the curvature has developed. The paraplegia is slow in its development; the patient at first feels weak in the legs or has disturbance of sensation, numbness, tingling, pins and needles. The girdle sensation may be marked, or severe pains in the course of the intercostal nerves. Motion is, as a rule, more quickly lost than sensation. Finally, there is complete interruption with the production of paraplegia, usually of the spastic type, with exaggeration of the reflexes. This may persist for months, or even for more than a year, and recovery still be possible.

(*c*) *Lumbar Region.*—In the lower dorsal and lumbar regions the symptoms are practically the same, but the sphincter centres are involved and the reflexes are not exaggerated.

Diagnosis.—Caries is by far the most frequent cause of slow compression of the cord, and when there are external signs the recognition is easy. There are cases in which the exudation in the spinal canal between the dura and the bone leads to compression before there are any signs of caries, and if the root symptoms are absent it may be extremely difficult to arrive at a diagnosis. Janeway has called attention to persistent lumbago as a symptom of importance in masked Pott's disease, particularly after injury. Brown-Séquard's paralysis is more common in tumor and in injuries than in caries. Pressure on the nerve-roots, too, is less frequent in caries than in malignant disease. The cervical form of pachymeningitis also produces a pressure paralysis, the symptoms of which have already been detailed. Pressure from cancer is naturally suggested when spinal symptoms follow within a few years after an operation. In paraplegia following tumor of the vertebra secondary to cancer of the breast, and in the erosion of the spine by retroperitoneal growths, the suffering is most intense. The condition has been well termed *paraplegia dolorosa.*

Treatment.—In compression by aneurism or tumor the condition is hopeless. In the former the pains are often not very severe, but in the latter morphia is always necessary. On the other hand, compression by caries is often successfully relieved even after the paralysis has persisted for a long period. When caries is recognized early, rest and support to the spine by the various methods now used by surgeons may do much to prevent the onset of paraplegia. When paralysis has developed, rest with extension gives the best hope of recovery. It is to be remembered that restoration may occur after compression of the cord has lasted for many months, or even more than a year. Cases have been cured by rest alone; the extradural and inflammatory products are absorbed and the caries heal. The most brilliant results in these cases have been obtained by suspension, a method introduced by J. K. Mitchell in 1826, and pursued with remarkable success by his son, Weir Mitchell. During my association with the Infirmary for Nervous Diseases I had numerous opportunities of witnessing the really remarkable effects of persistent suspension, even in apparently desperate and protracted cases. Mitchell's conclusions are that suspension should be employed early in Pott's disease; that used with care it enables us slowly to lessen the curve; that in these cases there must be, in some form, a replacement of the crumpled tissues; that unless there is great loss of power the use of the spine-car or chair of J. K. Mitchell enables suspension, especially in children, to be combined with some exercise; that no case of Pott's disease should be considered desperate without its trial; that suspension has succeeded after failures of other accepted methods; that the pull probably acts more or less directly on the cord itself, and that the gain is not explicable merely by obvious effects on the angular bony curve; that the methods of extension to be used in carious cases may be very varied, provided only we get active extension; that the plan and the length of time of extension must be made to conform to the needs, endurance, and sensation of the individual case. It may be months before there are any signs of improvement. In protracted cases, after suspension has been tried for months, laminectomy may be considered, and has in some instances been successful.*

The general treatment of caries is that of tuberculosis—fresh air, good food, cod-liver oil, and arsenic. Counter-irritation in these instances is of doubtful value.

Unilateral Lesions of the Spinal Cord (Brown-Séquard's Paralysis).—Tumors, stab wounds, and less frequently fracture or caries, may destroy one half of the cord, causing a peculiar and definite palsy, which was first recognized by Brown-Séquard, after whom it has been named. In a unilateral lesion the motor fibres are interrupted after their decussation in the medulla, consequently there is paralysis of the leg, or, if the lesion is in the cervical cord, of the arm and leg on the same side—spinal hemi-

* See full discussion of the subject by J. William White, Therapeutic Gazette, 1891.

plegia. As the sensory fibres, entering the cord through the posterior roots, decussate at once and ascend in the opposite half of the cord, there is loss of sensation on the side opposite to the lesion, so that in hemisection of the cervical cord above the brachial enlargement there is motor paralysis of the arm and leg of the same side and anæsthesia of the arm and leg of the opposite side. The anæsthesia may be only to painful and to thermic sensation. In many cases the tactile sensation is unimpaired. The muscular sense is diminished on the same side as the lesion, and on this side also the skin is hyperæsthetic, so that a slight irritation is felt very acutely. Of this phenomenon, which may persist for years, no satisfactory explanation has been given. Just above the level of the hyperæsthesia there is a narrow zone of anæsthesia, which is at the exact physiological level of the lesion and corresponds to the fibres coming from the same side, which are involved at once on entering the cord. Above this again there is a narrow zone of hyperæsthesia. The reflexes are usually increased on the side of the lesion and the temperature is slightly raised.

The following table of Gowers illustrates the distribution of these various symptoms in a hemi-lesion of the cord:

Cord.

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia.	Lesion.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened and then increased. Temperature raised.		Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

It is only in exceptional cases that all these features are met with in a case of Brown-Séquard's paralysis, and the condition may be transitory and rapidly replaced by paraplegia.

VI. Lesions of the Cauda Equina and Conus Medullaris.

The spinal cord extends only to the second lumbar vertebra. Injury, tumors, and caries at or below this level involve not the cord itself, but the bundle of nerves known as the cauda equina and the terminal portion of the cord, the conus medullaris. Much attention has been given recently to lesions of this part. The whole subject is admirably discussed in a recent work by Thorburn.* Fractures and dislocations are common in the lumbo-sacral region, tumors not infrequently involve the filaments of the cauda equina, and some of the nerves may be entangled in the cicatrix of a spina bifida.

* A Contribution to the Surgery of the Spinal Cord. By William Thorburn. London, 1890.

In a fracture or dislocation of the first lumbar vertebra the conus medullaris may be compressed with the last sacral nerves given off from it. In a case recently reported by Kirchhoff there was laceration of the conus with complete paralysis of the bladder and rectum, a case which is held to favor the view that the ano-vesical centre in man is situated in this region of the cord. There are several instances on record in which injury of the cauda equina has produced paralysis of the bladder and rectum alone, sometimes with a slight patch of anæsthesia in the neighborhood of the coccyx or the perinæum. More commonly branches of the sacral or lumbar nerve roots are involved producing an irregularly distributed motor and sensory paralysis in the legs. When the lumbar nerve-roots from the second to the fifth are compressed there is paralysis of the muscles of the legs, with the exception of the flexors of the ankles, the peronæi, the long flexors of the toes, and the intrinsic muscles of the feet, and loss of sensation in the front, inner, and outer part of the thighs, the inner side of the legs, and the inner side of the foot. The sacral roots may alone be involved. Thus in a case which I have reported the patient fell from a bridge and had paralysis of the legs and of the bladder and rectum. When seen sixteen years after the injury, there was slight weakness, with wasting of the left leg; there was complete loss of the function in the ano-vesical and genital centres, and anæsthesia in a strip at the back part of the thigh (in the distribution of the small sciatic), and of the perinæum, scrotum, and penis. The urethra was also insensitive. In a second case, in a young man with a healed spina bifida there was, with a small area of anæsthesia, involvement of the bladder and rectum, but retention of the sexual power.

Starr's table, given in the section on motor localization, will be found useful in determining the nerve fibres and segments involved in these cases of injury of the cauda equina.

VII. Tumors of the Spinal Cord and its Membranes.

New growths may develop in the cord or in its membranes, or may extend into them from the spine. The first two alone will be considered. Occasionally lipoma and parasites occur in the extradural space. Within the dura fibromata, sarcomata, and syphilitic and tuberculous growths are most common. In the cord itself, and attached to the pia mater, the tuberculous, syphilitic, and gliomatous growths are most frequent. Of 50 cases of tumor of the spinal cord and its envelopes analyzed by Mills and Lloyd, only 3 were parasitic. Of these 26 were some form of neoplasm, of which sarcomata were most common, 5 were gummatous, and 4 tuberculous. Herter has recently reported 3 cases of solitary tubercle in the cord, and has analyzed others from the literature. Of 24 cases in which the age was given, 15 occurred between the ages of fifteen and thirty-five, and 5 before the fifth year. The tumor is most common in

the dorsal and lumbar regions, and is usually met with in connection with tuberculous lesions elsewhere.

The anatomical effects of tumors are very varied. Slow compression is usually produced by growths external to the cord, and it is remarkable what a high grade of compression the cord will bear without serious interference with its functions. In cases of prolonged interruption ascending and descending degenerations occur. Tumors developing within the cord may lead to syringo-myelia. And, lastly, tumors not infrequently excite intense myelitis.

Symptoms.—These will naturally vary a good deal with the segment involved and with the degree of pressure and the extent of implication of the nerve-roots.

Within the cord the symptoms are those of a gradually progressing paraplegia, which may at first have the picture of a Brown-Séquard paralysis. Atrophy follows the involvement of the anterior cornua, and vasomotor disturbances may be marked. The reflexes are lost at the level of the lesion, but if in the dorsal cord, the reflexes are retained in the legs. The symptoms are apt to be complicated with those of acute or subacute myelitis, which may completely alter the clinical picture. Tumors of the spinal membranes are characterized by the early onset and persistence of the root symptoms, which consist of radiating pains, girdle sensation, hyperæsthesia, or anæsthesia in various portions of the trunk. There may even be severe pain in the anæsthetic areas. Irritation of the motor roots may cause spasm of the muscles supplied, or wasting with paralysis. The paraplegia supervenes some time after the occurrence of the root symptoms. In the dorsal region the level of the growth is usually accurately defined by the level of the pain and the condition of the reflexes.

The diagnosis of tumor within the cord is sometimes easy, the characteristic features being the constancy and severity of the root symptoms at the level of the growth and the progressive paralysis. Caries may cause identical symptoms, but the radiating pains are rarely so severe. Cervical meningitis simulates tumor very closely, and in reality produces identical effects, but the very slow progress and the bilateral character from the outset may be sufficient to distinguish this.

In chronic transverse myelitis the symptoms may, according to Gowers, simulate tumor very closely and present radiating pains, a sense of constriction, and progressive paralysis.

The nature of the tumor can rarely be indicated with precision. With a marked syphilitic history gumma may naturally be suspected, and with coexisting tuberculous disease a solitary tubercle.

Treatment.—If the possibility of syphilitic infection is present the iodide of potassium should be given in large and increasing doses. For the severe pains counter-irritation is sometimes beneficial, particularly the thermo-cautery; morphia is, however, often necessary.

In a few instances tumors of the cord or of the membranes are amena-

Abscess of the cord is a rare lesion of which only three or four cases have been described, all metastatic. It may occur without meningitis

(Rev. Neur. 1893. 4)

ble to surgical treatment. The removal by Victor Horsley of a growth from the membranes of the cord in a patient of Gowers' was one of the most brilliant operations of modern surgery.

VIII. Progressive (Spinal) Muscular Atrophy

(*Chronic Degeneration of the Motor Nuclei—Poliomyelitis Anterior Chronica*).

Definition.—A disease characterized by degeneration of groups of the motor nuclei in the cord and medulla, with wasting of the corresponding muscles. The pyramidal tracts are usually involved, and the paralysis may have a spastic character. In some cases the degeneration has been traced to the ganglion cells of the motor cortex.

Three affections, as a rule described apart, belong together in this category: (*a*) Progressive muscular atrophy of spinal origin; (*b*) amyotrophic lateral sclerosis; and (*c*) progressive bulbar paralysis. A slow atrophic change in the motor nuclei is the anatomical basis, and the disease, as Charcot states, is one of the whole motor path, involving, in many cases, the cortical, bulbar, and spinal centres. There may be simple muscular atrophy with little or no spasm, or progressive wasting with marked spasm and great increase in the reflexes. In others, there are added symptoms of involvement of the motor nuclei in the medulla—a glosso-labio-laryngeal paralysis; while in others, again, with atrophy (especially of the arms), a spastic condition of the legs, and bulbar phenomena, tremors develop and signs of cortical lesion. These various stages may be traced in the same case. I have for ten years had under observation a man whose illness began with weakness and atrophy of the hand muscles. Gradually the legs began to get stiff and the gait spastic; the arms subsequently wasted and the reflexes were increased. After these symptoms had persisted with increasing intensity for six or seven years, certain of the motor nuclei of the medulla became involved, the speech became thick, and the movements of the lips and tongue were impaired. Tremor has developed of late in the arms and hands. With these chronic changes the visceral functions have remained unimpaired and the mind unaffected. It has been a lesion of the motor segments, beginning in the lower and gradually extending upward. The disease began as progressive atrophy, and gradually assumed a typical picture of amyotrophic lateral sclerosis, and now the bulbar features are well marked and the tremor would indicate that the cortex is also involved.

For convenience, bulbar paralysis will be considered separately, and I shall here take up together *progressive muscular atrophy* and *amyotrophic lateral sclerosis*.

The disease is known as the Aran-Duchenne type of progressive muscular atrophy, after the French physicians who early described it, and as Cruveilhier's palsy. Lockhard Clarke demonstrated that it was a spinal

lesion. Charcot separated the two types—one with simple wasting, in which the anterior horns are alone involved; and the other in which, with degeneration of the cornua, the pyramidal tracts are affected, causing wasting *plus* a spastic condition. To this he gave the name of amyotrophic lateral sclerosis. There is but little evidence, however, to show that the anterior horns are ever affected without secondary changes in the pyramidal tracts, and Leyden and Gowers regard the two diseases as identical.

Etiology.—The cause of the disease is unknown. It is more frequent in males than in females. It attacks adults, developing after the thirtieth year, though occasionally younger persons are attacked. A large majority of all cases of progressive muscular atrophy under twenty-five years of age are of myopathic (i. e., muscular), not myelopathic (i. e., spinal) origin. Cold, wet, exposure, fright, and mental worries are mentioned as possible causes. Hereditary influences are present in certain cases. The father of the man whose case is referred to above died of progressive wasting of the muscles, but there have been no other cases in the family. It is highly probable that when many members of a family are affected the disease is not spinal, but an idiopathic muscular atrophy; and yet, in the Farr family, which I recorded a few years ago, in which thirteen members were affected in two generations, with the exception of two, the cases occurred or proved fatal above the age of forty, and the late onset speaks rather for a spinal affection. The amyotrophic form may develop late in life—after seventy—as a senile change.

Morbid Anatomy.—The following are the important anatomical changes: (*a*) The muscles waste and undergo fatty and sclerotic changes. The terminal branches of the motor nerves are degenerated. (*b*) The anterior roots are atrophied in those sections of the cord corresponding to the wasted muscles. (*c*) The gray matter shows the most marked alteration. The large ganglion cells of the anterior horns are atrophied, or, in places, have entirely disappeared, the neurogliar tissue is increased, and the fibres of the anterior nerve-root passing through the white matter are wasted. (*d*) In a majority of all the cases there is sclerosis in the antero-lateral tracts, but the direct cerebellar and the antero-lateral ascending tracts are spared. It was to this combination of atrophy of the anterior horns and sclerosis of the antero-lateral columns that Charcot gave the name amyotrophic lateral sclerosis. (*e*) The degeneration of the gray matter is rarely confined to the cord, but extends to the medulla; the motor nuclei are found extensively wasted in cases which have shown bulbar symptoms during life. (*f*) Cerebral changes also occur. The pyramidal tracts have been found degenerated through the pons and capsule, and in the motor cortex the large ganglion cells are wasted.

The essential anatomical change is a slow degeneration of the motor path, involving specially the nerve-cells of the anterior cornua and the anterior root-fibres, to which the loss of power and wasting in the muscles

Not only may the jaw reflex be present but it is one of the rare conditions in which a jaw clonus may be obtained

are secondary. The upper segment is also involved, either simultaneously or at a later period.

Symptoms.—Irregular pains may precede the onset of the wasting. In one case the pains were about the hip and shoulder joints and the patient was treated for chronic rheumatism. The hands are first affected, and there is difficulty in performing delicate manipulations. The muscles of the ball of the thumb waste early, then the interossei and lumbricales, leaving marked depressions between the metacarpal bones. Ultimately the contraction of the flexor and extensor muscles and the extreme atrophy of the thumb muscles, the interossei, and lumbricales produces the claw-hand—*main en griffe* of Duchenne. The flexors of the forearm are usually involved before the extensors. In the shoulder-girdle the deltoid wastes first; it may waste even before the other muscles of the upper extremity. The trunk muscles are gradually attacked; the upper part of the trapezius long remains unaffected. Owing to the feebleness of the muscles which support it, the head tends to fall forward. The platysma myoides is unaffected and often hypertrophies. The arms and the trunk muscles may be much atrophied before the legs are attacked. The glutei, the vasti, and the tibialis anticus are first attacked when the disease begins in the legs. In the member of the Farr family who came under my notice (if this was really a myelopathic disorder) the wasting began in the gluteal and hamstring muscles of the left leg. The face muscles are attacked late. Ultimately the intercostal and abdominal muscles may be involved, the wasting proceeds to an extreme grade, and the patient may be actually "skin and bone," and, as "living skeletons," the cases are not uncommon in "museums" and "side-shows." Deformities and contractures result, and lordosis is almost always present. A curious twitching of the muscles (fibrillation) is a common symptom, and may occur in muscles which are not yet attacked. It is not, as was formerly supposed, a characteristic feature of the disease. The irritability of the muscle is increased. Sensation is unimpaired, but the patient may complain of numbness and coldness of the affected limbs. The galvanic and faradic irritability of the muscles progressively diminishes and may become extinct, the galvanic persisting for the longest time. In cases of rapid wasting and paralysis there may be the reaction of degeneration. The excitability of the nerve-trunks may persist after the muscles have ceased to respond. The loss of power is usually proportionate to the degree of wasting.

The foregoing description applies to the group of cases in which the atrophy and paralysis are flaccid—*atonic*, as Gowers calls it. In other cases, those which Charcot describes as amyotrophic lateral sclerosis, with the wasting there is more or less spasm, which may exist from the outset. This *tonic* atrophy may involve the legs chiefly or is present in the arms and legs. The reflexes are greatly increased. The most typical condition of spastic paraplegia may be produced. On starting to walk, the patient

seems glued to the ground and makes ineffectual attempts to lift the toes; then four or five short, quick steps are taken on the toes with the body thrown forward; and finally he starts off, sometimes with great rapidity. Some of the patients can walk up and down stairs better than on the level. The wasting is never so extreme as in the atonic form, and the loss of power may be out of proportion to it. The sphincters are unaffected. Sexual power may be lost early.

As the degeneration extends upward an important change takes place from the development of bulbar symptoms, which may, however, precede the spinal manifestations. The lips, tongue, face, pharynx, and larynx may be involved. The lips may be affected and articulation impaired for years before serious symptoms occur. In the final stage there may be tremor, the memory fails, and a condition of dementia may develop.

Gowers gives the following useful classification of the varieties of this affection: (1) Atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting or with only slight wasting; and (3) atonic atrophy, rarely extreme in degree, with excess of the reflexes. These conditions may "coexist in every degree and combination—between universal atonic atrophy on the one hand and universal spastic paralysis without wasting on the other."

Diagnosis.—The affection must be distinguished from the primary muscular atrophies which usually occur in younger persons, often affect many members of a family, and have a different distribution, beginning either in the muscles of the shoulder girdle—sparing the hands or involving the face and upper-arm muscles—or the peroneal group. Muscular atrophy in the adult, beginning in the muscles of the thumbs, gradually involving the interossei and lumbricales, as a rule is of myelopathic origin.

Treatment.—The disease is incurable. I have never seen the slightest benefit from drugs or electricity. The downward progress is slow but certain, though in a few cases a temporary arrest may take place. With a history of syphilis, mercury and iodide of potassium may be tried, and Gowers recommends courses of arsenic and strychnine. Probably the most useful means is systematic massage, particularly in the spastic cases.

Bulbar Paralysis (*Glosso-labio-laryngeal Paralysis*).

An affection of the motor nuclei of the medulla oblongata, rarely primary, more commonly a part of a general degenerative affection of the nuclei of the motor path. The disease is sometimes called by the name of Duchenne. Acute and chronic forms may be recognized.

(1) *Acute bulbar paralysis* may be due to (*a*) hæmorrhagic or embolic softening in the pons and medulla; (*b*) acute inflammatory softening, analogous to polio-myelitis, occurring occasionally as a post-febrile affection.

The onset is usually sudden, hence the term apoplectiform. The cases

are almost invariably bilateral. As the nuclei presiding over the muscles of the tongue and lips are involved the speech is almost or entirely lost. The saliva drools, the lips are flabby and flaccid, swallowing may be difficult, and there may be loss of power in the laryngeal muscles. Usually these cases rapidly prove fatal, but occasionally a case with a sudden onset, like that figured by Gowers, may become chronic. In these acute cases there may be loss of power in one arm, or hemiplegia, sometimes alternate hemiplegia, with paralysis on one side of the face and loss of power on the other side of the body.

(2) *Chronic bulbar paralysis* is an affection of adult life, rarely beginning under the fortieth year, and in a great majority of the cases it is only part of a general degeneration of the motor nuclei. The disease usually begins with slight defect in the speech, and the patient has difficulty in pronouncing the dentals and linguals. The paralysis starts in the tongue, and the superior lingual muscle gradually becomes atrophied, and finally the mucous membrane is thrown into transverse folds. In the process of wasting the fibrillary tremors are seen. Owing to the loss of power in the tongue, the food is with difficulty pushed back into the pharynx. The saliva also may be increased, and is apt to accumulate in the mouth. When the lips become involved the patient can neither whistle nor pronounce the vowels *o* and *u*. The mouth looks large, the lips are prominent, and there is constant drooling. The food is masticated with difficulty. Swallowing becomes difficult, owing partly to the regurgitation into the nostrils, partly to the involvement of the pharyngeal muscles. The muscles of the vocal cords waste and the voice becomes feeble, but the laryngeal paralysis is rarely so extreme as that of the lips and tongue.

The course of the disease is slow but progressive. Death often results from an aspiration pneumonia, sometimes from choking, more rarely from involvement of the respiratory centres. The mind usually remains clear. The patient may become emotional. In a majority of the cases the disease is only part of a progressive atrophy, either simple or associated with a spastic condition. In the latter stage of amyotrophic lateral sclerosis the bulbar lesions may paralyze the lips long before the pharynx or larynx becomes affected.

The *diagnosis* of the disease is readily made, either in the acute or chronic form. The involvement of the lips and tongue is usually well marked, while that of the palate may be long deferred. A condition has been described, however, which may closely simulate bulbar paralysis. This is the so-called *pseudo-bulbar* form or bulbar palsy of cerebral origin. Bilateral disease of the motor cortex in the lower part of the ascending frontal convolution may cause paralysis of the lips and tongue and pharynx, which closely simulates a lesion of the medulla. Sometimes the symptoms appear on one side, but in many instances they develop suddenly on both sides. A bilateral lesion has usually been found, but in several instances the disease was unilateral.

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Progressive bulbar paralysis is an incurable affection. Transient improvement may occur. Strychnine may be tried. Electricity is of doubtful benefit. Special care must be taken in feeding these patients, and when deglutition becomes much impaired the stomach-tube should be employed.

III. DISEASES OF THE BRAIN.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater (*Pachymeningitis*).

(*a*) **Pachymeningitis Externa.**—Hæmorrhage often occurs as a result of fracture. Inflammation of the external layer of the dura is rare. Caries of the bone, either extension from middle-ear disease or due to syphilis, is the principal cause. In the syphilitic cases there may be a great thickening of the inner table and a large collection of pus between the dura and the bone. In a remarkable case of this kind at the Montreal General Hospital the frontal lobes were so compressed by the thickened skull, and the purulent effusion between the bone and the dura, that the anterior vertical measurement of the brain was only 2·5 cm., while that of the posterior part was 8 cm.

Occasionally the pus is infiltrated between the two layers of the dura mater or may extend through and cause a dura-arachnitis.

The symptoms of external pachymeningitis are indefinite. In the syphilitic cases there may be a small sinus communicating with the exterior. Compression symptoms may occur with or without paralysis.

(*b*) **Pachymeningitis Interna.**—This occurs in three forms: (1) Pseudo-membranous, (2) purulent, and (3) hæmorrhagic. The first two are unimportant. Pseudo-membranous inflammation of the lining membrane of the dura is not usually recognized, but a most characteristic example of it came under my observation as a secondary process in pneumonia. Purulent pachymeningitis may follow an injury, but is more commonly the result of extension from inflammation of the pia. It is remarkable how rarely pus is found between the dura and arachnoid membranes.

Hæmorrhagic Pachymeningitis (*Hæmatoma of the Dura Mater*).

This remarkable condition, first described by Virchow, is very rare in general medical practice. During ten years no instance of it came under my observation at the Montreal General Hospital. On the other hand, in the post-mortem room of the Philadelphia Hospital, which received material from a large almshouse and asylum, the cases were not uncommon, and within three months I saw four characteristic examples, three of which came from the medical wards. On the other hand, the frequency

of the condition in asylum work may be gathered from the fact that Wigglesworth found 42 examples in a series of 400 unselected post-mortem examinations.

The disease is found chiefly in males and in persons over fifty years of age. It is most frequent in forms of chronic insanity and in chronic alcoholism. It has also been found in profound anæmia and other blood conditions, and is said to have followed certain of the acute fevers.

The morbid anatomy is interesting. Virchow's view that the delicate vascular membrane precedes the hæmorrhage is undoubtedly correct. Practically we see one of three conditions in these cases: (*a*) Subdural vascular membranes, often of extreme delicacy; (*b*) simple subdural hæmorrhage; (*c*) combination of the two, vascular membrane and blood-clot. Certainly the vascular membrane may exist without a trace of hæmorrhage —simply a fibrous sheet of varying thickness, permeated with large vessels, which may form beautiful arborescent tufts. On the other hand, there are instances in which the subdural hæmorrhage is found alone—in 15 out of Wigglesworth's 42 cases—but it is possible that in some of these at least the hæmorrhage may have destroyed all trace of the vascular membrane. In some cases a series of laminated clots are found, forming a layer from 3 to 5 mm. in thickness. Cysts may occur within this membrane. The source of the hæmorrhage is probably the dural vessels. Hugenin and others hold that the bleeding comes from the vessels of the pia mater, but certainly in the early stage of the condition there is no evidence of this; on the other hand, the highly vascular subdural membrane may be seen covered with the thinnest possible sheeting of clot, which has evidently come from the dura. The subdural hæmorrhage is usually associated with atrophy of the convolutions, and it is held that this is one reason why it is so common in the insane; but there must be some other factor than atrophy, or we should meet with it in phthisis and various cachectic conditions in which the cerebral wasting is as common and almost as marked as in cases of insanity.

The symptoms are indefinite, and the diagnosis cannot be made with certainty. Headache has been a prominent symptom in some cases, and when the condition exists on one side there may be hemiplegia. Extensive bilateral disease may exist without any symptoms whatever.

Diseases of the Pia Mater.

(*a*) **Acute Leptomeningitis.**—In this form the exudation is between the pia and the arachnoid membranes.

Etiology.—Acute inflammation of the pia mater occurs under the following circumstances: (1) As a result of an eruption of tubercles, most frequently in the basal meninges, forming the basilar or tuberculous meningitis which has been already considered (see tuberculosis). (2) In the epidemic cerebro-spinal fever. (3) Secondary to acute general diseases,

more particularly pneumonia, less frequently small-pox, typhoid fever, rheumatic fever, whooping cough, scarlet fever, and measles. In erysipelas meningitis may arise either by infection through the blood or by direct extension. Cases in which the inflammation passes through the bone are extremely rare; on the other hand, there are instances of extensive erysipelas of the face in which the disease travels along the nerve-roots and so reaches the meninges. In this group pneumonia is the only disease which is frequently followed by meningitis. In one hundred autopsies at the Montreal General Hospital in pneumonia, meningitis was found eight times, and I had several opportunities of seeing cases of similar character in Philadelphia. In septicæmia and pyæmia, including ulcerative endocarditis in this category, acute meningitis is not very rare. In ulcerative endocarditis it is common, as may be judged from the statistics which I collected of 209 cases, of which 25 were complicated with meningitis. No instance has fallen under my observation in connection with typhoid fever or rheumatic fever.

(4) Injury or disease of the bones of the skull, perforating wounds of the orbit, or as a sequence of abscess which is the result of injury. Under this section by far the most frequent cause is necrosis in the petrous portion of the temporal bone, which may excite either extensive inflammation of the pia mater or abscess of the brain. (5) In certain constitutional conditions, such as gout and Bright's disease. This form is usually basilar and comes on insidiously. Gout is usually mentioned as a cause of meningitis, but it must be extremely rare. Duckworth does not refer to it in his work, and the symptoms of the so-called cerebral gout can scarcely be separated from those of uræmia. On the other hand, in Bright's disease, I have met with at least three instances of well-marked meningitis, chiefly of the base.

(6) While in a great majority of all cases of basilar meningitis in children tubercles may be found, a simple *leptomeningitis infantum* must also be recognized. Cases are not very uncommon. Two occurred in debilitated children under my care at the Infants' Home in Montreal, and I saw at least two specimens of the kind at the Philadelphia Hospital. The condition may be limited to the meninges at the base, particularly at the posterior part, and to the under surface of the cerebellum. It has also been termed occlusive meningitis, owing to the fact that involving chiefly the posterior portion of the meninges about the cerebellum and medulla, the foramen of Magendie may be closed, with the result of acute, sometimes purulent hydrocephalus, as described by Gee and Barlow.* (7) Other causes mentioned are sun-stroke and excessive study, which are probably doubtful. Syphilis, which is a common cause of chronic meningitis, rarely induces the acute form.

Morbid Anatomy.—The basal or cortical meninges may be involved. In the form associated with pneumonia and ulcerative endocarditis the

* On the Cervical Opisthotonos of Infants, St. Bartholomew's Hospital Reports, 1878.

Meningitis serosa. (Lumbale.

Child ät. 5. admitted to my wards. H. died June 10 ät.
Comatose retracted head. dilated pupils. grinding of teeth. Illness
of 2 weeks duration, great emaciation. Broncho-pneumonia
of left lower lobe; diagnosis of tuberculous meningitis. autopsy. no
tubercles in lungs. Gt distension of lateral & third ventricles
& fluid - a little turbid. Ependyma fornix, not ulcerated
no tubercles. Adhesions form & choroid plexuses.

disease is bilateral and usually limited to the cortex. In extension from disease of the ear it is usually unilateral and may be accompanied with abscess or with thrombosis of the sinuses. In the non-tuberculous form in children, in the meningitis of chronic Bright's disease, and in cachectic conditions the base is usually involved. The vessels are injected, the subarachnoid fluid is increased and becomes opaque. The arachnoid is also turbid, and there may be a yellowish-white, creamy exudate, or a grayish-green purulent matter beneath the arachnoid. The interpeduncular space may be completely filled with the exudate, which extends upon the under surface of the cerebellum. In the cases secondary to pneumonia the effusion beneath the arachnoid may be very thick and purulent, completely hiding the convolutions. The ventricles also may be involved, though in these simple forms they rarely present the distention and softening which is so frequent in the tuberculous meningitis.

The *leptomeningitis infantum* may present a picture very similar to the tuberculous disease. There is exudation about the optic chiasma and in the Sylvian fissures and toward the cerebellum. In some instances we can say definitely that the condition is not tuberculous only after the most careful search in the meninges and central arteries, and when no tubercles are found in the lungs and bronchial glands. In other instances the meningitis may be limited to the posterior part of the base, about the pons, cerebellum, and fourth ventricle, and the lateral ventricles may present a most remarkable ependymitis. In a specimen recently shown to me by W. T. Howard, Jr., from a child aged three months (which had had an operation performed for imperforate anus), there was posterior basilar meningitis, the fourth ventricle was filled with pus, the walls thickened, rough, and infiltrated with pus; the lateral ventricles were enormously distended with pus, and the ependyma, which was from two to three millimetres in diameter, was softened and in a condition of purulent infiltration. A coccus and the *bacterium coli commune* were found in the pus. In a somewhat similar case at the Philadelphia Hospital the ependymitis was limited to the posterior and descending cornua, which were greatly distended and contained pus. The anterior cornua were little, if at all, affected, owing doubtless to the influence of gravity. This condition of intense purulent ependymitis is rare in the adult, but I remember to have seen an instance of it in a patient of Pepper's at the University Hospital, Philadelphia.

Symptoms.—I have already spoken at length of the clinical features of tuberculous meningitis, which is by far the most common and important form. The other varieties have a general resemblance to it, particularly those in which the base is affected. I have already, on several occasions, called attention to the fact that cortical meningitis is not to be recognized by any symptoms or set of symptoms from a condition which may be produced by the poison of many of the specific fevers. In the cases of so-called cerebral pneumonia, unless the base is involved and the

nerves affected, the disease is unrecognizable, since identical symptoms may be produced by intense engorgement of the meninges. In typhoid fever, in which meningitis is very rare, the twitchings, spasms, and retractions of the neck are almost invariably associated with cerebro-spinal congestion, not with meningitis.

A knowledge of the etiology gives a very important clew. Thus, in middle-ear disease the development of high fever, delirium, vomiting, convulsions, and retraction of the head and neck would be extremely suggestive of meningitis or abscess. Headache, which may be severe and continuous, is the most common symptom. In the fevers, particularly in pneumonia, there may be no complaint of headache. Delirium is frequently early, and is most marked when the fever is high. Convulsions are less common in simple than in tuberculous meningitis. They were not present in a single instance in the cases which I have seen in pneumonia, ulcerative endocarditis, or septicæmia. In the simple meningitis of children they may occur. Rigidity and spasm or twitchings of the muscles are more common. Stiffness and retraction of the muscles of the neck are important symptoms; but they are by no means constant, and are most frequent when the inflammation extends to the meninges of the cervical cord. Vomiting is a common symptom in the early stages, particularly in basilar meningitis. Constipation is usually present. Optic neuritis is rare in the meningitis of the cortex, but is not uncommon when the base is involved.

Important symptoms are due to lesions of the nerves at the base. Strabismus or ptosis may occur. The facial nerve may be involved, producing slight paralysis, or there may be damage to the fifth nerve, producing anæsthesia and, if the Gasserian ganglion is affected, trophic changes in the cornea. The pupils are at first contracted, subsequently dilated, and perhaps unequal.

Fever is present, moderate in grade, rarely rising above 103°. In the non-tuberculous leptomeningitis of debilitated children and in Bright's disease there may be little or no fever. The pulse may be increased in frequency at first and subsequently is slow and irregular.

Treatment.—There are no remedies which in any way control the course of acute meningitis. An ice-bag should be applied to the head and, if the subject is young and full-blooded, general or local depletion may be practised. Absolute rest and quiet should be enjoined. When disease of the ear is present, a surgeon should be early called in consultation, and if there are symptoms of meningo-encephalitis which can in any way be localized trephining should be practised. An occasional saline purge will do more to relieve the congestion than blisters and local depletion. I have no belief whatever in the efficacy of counter-irritation to the back of the neck, and to apply a blister to a patient suffering with agonizing headache in meningitis is needlessly to add to the suffering. If counter-irritation is deemed essential, the thermo-cau-

A Draperies. of this nature & the case symptoms which
Saundby mentions (see Powers)

Quincke's lumbar puncture (see p.) has been used with success by Fürbringer. Forty cc of cloudy fluid were removed in which tubercle bacilli were found. The headache & other cerebral symptoms disappeared and the patient, a man of 20, recovered. Wallis Ord & Underhume report a case of recovery, in a child of 5 years, after trephining & drainage of Lark sp.

tery, lightly applied, is more satisfactory, because the pain inflicted is transient.

The gastro-intestinal symptoms should receive appropriate treatment. Gowers states that in two instances of septic meningitis which recovered the good effects seemed to be due to large doses of the perchloride of iron. Iodide of potassium and mercury are recommended by some authors.

The application of an ice-cap, attention to the bowels and stomach, and keeping the fever at a moderate height by sponging, are the necessary measures in a disease recognized as almost invariably fatal, and in which the cases of recovery are extremely doubtful.

(*b*) *Chronic Leptomeningitis.*—This is rarely seen apart from syphilis or tuberculosis, in which the meningitis is associated with the growth of the granulomata in the meninges and about the vessels. The symptoms in such cases are extremely variable, depending entirely upon the situation of the growth. They may closely resemble those of tumor and be associated with localized convulsions. The leptomeningitis infantum may be chronic. In the cases reported by Gee and Barlow the duration in some instances extended even to a year and a half. The involvement of the posterior part of the meninges and of the ventricles may lead to dilatation and hydrocephalus. The symptoms upon which these authors lay stress are convulsions, and retraction of the head, which is particularly marked when the child is made to sit up. There may be rigidity of the limbs and epileptiform convulsions.

II. AFFECTIONS OF THE BLOOD-VESSELS.

Hyperæmia.

Congestion of the brain has played an important part in cerebral pathology. Undoubtedly there are great variations in the amount of blood in the cerebral vessels; this is universally conceded, but how far these changes are associated with a definite group of symptoms is not quite so clear. The hyperæmia may be either active or passive.

Active hyperæmia is associated with febrile conditions, with increased action of the heart, chilling of the surface, contraction of the superficial vessels, and with the suppression of certain customary discharges. Among other recognized causes are plethora, functional irritation, such as is associated with excessive brain work, and the action of certain substances, such as alcohol and nitrite of amyl.

Passive hyperæmia results from obstruction in the cerebral sinuses and veins, engorgement in the lesser circulation, as in mitral stenosis, emphysema, from pressure on the superior cava by aneurisms and tumors, and in the venous engorgement which takes place in prolonged straining

efforts. In its most intense form it is seen in the compression of the superior cava by tumors and in death from strangulation.

The anatomical changes in congestion of the brain are by no means striking. Active hyperæmia is never visible post mortem. The veins of the cortex are distended, the gray matter has a deeper color, and its vessels are full. The arteries at the base and in the Sylvian fissures contain blood. Nothing, however, can be more uncertain or indefinite than the post-mortem appearances of hyperæmia of the brain. The most intense distention of the vessels is seen in early death during the specific fevers, or in the secondary passive congestion due to obstruction in the superior cava or in the lesser circulation.

Symptoms.—There are no characteristic or constant features of cerebral hyperæmia. It may exist in the most extreme grade without the slightest disturbance of the cerebral functions, as is witnessed frequently in the pressure of tumors on the superior vena cava. How far the headache and delirium of the early stage of the infectious fevers is to be assigned to hyperæmia of the blood-vessels of the brain it is not easy to determine. The headache, dizziness, and unpleasant sensations in aortic insufficiency and in some instances of hypertrophy of the heart may be due to the cerebral congestion.

As a separate clinical entity, congestion of the brain rarely comes under observation. I have no knowledge of instances associated with delirium, fever, insomnia, and convulsions, or of the so-called apoplectiform variety described by some writers. Very plethoric persons are subject to attacks of headache with flushing of the face and irritability of temper, attacks which may recur frequently and are sometimes relieved by bleeding at the nose. These are usually attributed to congestion of the brain. When passive hyperæmia reaches a high grade, there may be torpor, dulness of the intellect, and ultimately deep coma.

Anæmia.

This may be induced by loss of blood, either quickly, as in hæmorrhage, or gradually, as in the severe primary and secondary anæmias. The anæmia may be local and due to causes which interfere with the blood supply to the brain, as narrowing of the vessels by endarteritis, pressure, narrowing of the aortic orifice, or it may follow an unequal distribution of the blood in consequence of dilatation of certain vascular territories. Thus, rapid distention of the intestinal vessels, such as occurs after the removal of ascitic fluid, may cause sudden death from cerebral anæmia. The commonest illustration of this is the fainting fit from emotion, in which the blood supply to the brain is insufficient on account of the diminished arterial pressure. Anæmia of the cerebral vessels may be caused by pressure of fluid in the ventricles. The partial anæmia results from obliteration of branches of the circle of Willis by embolism or throm-

bosis. Ligature of one carotid sometimes causes a transient marked anæmia and disturbance of function on one side of the brain.

The anatomical condition of the brain in anæmia is very striking. The membranes are pale, only the large veins are full, the small vessels over the gyri are empty, and an unusual amount of cerebro-spinal fluid is present. On section both the gray and white matter look extremely pale and the cut surface is moist. Very few *puncta vasculosa* are seen.

Symptoms.—The effects of anæmia of the brain are well illustrated by a fainting fit in which loss of consciousness follows the heart weakness. When the result of hæmorrhage, there are drowsiness, giddiness, inability to stand, flashes of light, and noises in the ear; the respiration becomes hurried; the skin is cool and covered with sweat; and gradually, if the bleeding continues, consciousness is lost and death may occur with convulsions. In ordinary syncope the loss of consciousness is usually transient and the recumbent posture alone may suffice to restore the patient to consciousness. In the more chronic forms of brain anæmia, such as result from the gradual impoverishment of the blood, as in protracted illness or in starvation, the condition known as irritable weakness results. Mental effort is difficult, the slightest irritation is followed by undue excitement, the patient complains of giddiness and noises in the ears, or there may be hallucinations or delirium. These symptoms are met with in an extreme grade as a result of prolonged starvation.

An interesting set of symptoms, to which the term *hydrocephaloid* was applied by Marshall Hall, occurs in the debility produced by prolonged diarrhœa in children. The child is in a semi-comatose condition with the eyes open, the pupils contracted, and the fontanelle depressed. In the earlier period there may be convulsions. The coma may gradually deepen, the pupils become dilated, and there may be strabismus and even retraction of the head, symptoms which closely simulate basilar meningitis.

Œdema of the Brain.

In the pathology of brain lesions œdema formerly played a *rôle* almost equal in importance to congestion. It occurs under the following conditions: In general atrophy of the convolutions, in which case the œdema is represented by an increase in the cerebro-spinal fluid and in that of the meshes of the pia. In extreme hyperæmia from obstruction, as in mitral stenosis or in tumors, there may be a condition of congestive œdema, in which, in addition to great filling of the blood-vessels, the substance of the brain itself is unusually moist. The most acute œdema is a local process found around tumors and abscesses. An intense infiltration, local or general, may occur in Bright's disease, and to it, as Traube suggested, certain of the uræmic symptoms may be due.

The *anatomical changes* are not unlike those of anæmia. When a sequence of progressive atrophy, the fluid is chiefly within and beneath

the membranes. The brain substance is anæmic and moist, and has a wet, glistening appearance, which is very characteristic. In some instances the œdema is more intense and local and the brain substance may look infiltrated with fluid. The amount of fluid in the ventricles is usually increased.

The *symptoms* are in great part those of anæmia, and are not well defined. As just stated, some of the cerebral features of uræmia may depend upon it. Of late years cases have been reported by Raymond, Tenneson, and Dercum, in which unilateral convulsions or paralysis have occurred in connection with chronic Bright's disease, and in which the condition appeared to be associated with œdema of the brain. The older writers laid great stress upon an apoplexia serosa, which may really have been a general œdema of the brain.

Cerebral Hæmorrhage.

The bleeding may come from branches of either of the two great groups of cerebral vessels—the *basal*, comprising the circle of Willis and the central arteries passing from it, or the *cortical group*, the anterior, middle, and the posterior cerebral vessels. In a majority of the cases the hæmorrhage is from the central branches, more particularly from those given off by the middle cerebral arteries in the anterior perforated spaces, and which supply the corpora striata and internal capsules. One of the largest of these branches which passes to the third division of the lenticular nucleus and to the hinder part of the internal capsule is so frequently involved in hæmorrhage that it has been called by Charcot *the artery of cerebral hæmorrhage.* The bleeding may be into the substance of the brain, to which alone the term cerebral apoplexy is applied, or into the membranes, in which case it is termed meningeal hæmorrhage; both, however, are usually included under the terms intracranial or cerebral hæmorrhage.

Etiology.—The conditions which produce lesions of the blood-vessels play a very important part; thus the natural tendency to degeneration of the vessels in advanced life makes apoplexy much more common after the fiftieth year. It may, however, occur in children under ten. On account of the greater liability to arterial disease (associated probably with muscular exertion and the abuse of alcohol), men are more subject to cerebral hæmorrhage than women. Heredity was formerly thought to be an important factor in this affection, and the apoplectic *habitus* or build is still referred to. By this is meant a stout, plethoric body of medium size, with a short neck. Heredity influences cerebral hæmorrhage entirely through the arteries, and there are families in which they degenerate early, usually in association with renal changes. The secondary hypertrophy of the heart brings with it serious dangers, which have already been discussed in the section upon arteries. The three special factors in

inducing arterio-sclerosis—the abuse of alcohol, syphilis, and prolonged muscular exertion—are found to be important antecedents in a large number of cases of cerebral hæmorrhage.

The endocarditis of rheumatism and other fevers may indirectly lead to apoplexy by causing embolism and aneurism of the vessels of the brain. Cerebral hæmorrhage occurs occasionally in the specific fevers and in profound alterations of the blood, as in leukæmia and pernicious anæmia. The actual exciting cause of the hæmorrhage is not evident in the majority of cases. The attack may be sudden and without any preliminary symptoms. In other instances violent exertion, particularly straining efforts or the excited action of the heart in emotion may cause a rupture.

Morbid Anatomy.—The lesions causing apoplexy are almost invariably in the cerebral arteries, in which the following changes may lead directly to it:

(*a*) Periarteritis with the production of miliary aneurisms, rupture of which is the most common cause of cerebral hæmorrhage. They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. On section of the brain substance they may be seen as localized, small dark bodies about the size of a pin's head. Sometimes they are seen in numbers upon the arteries carefully withdrawn from the anterior perforated spaces. According to Charcot and Bouchard, who have described them, they are most frequent in the central ganglia. In apoplexy after the fortieth year if sought for they are rarely missed.

(*b*) Aneurism of the branches of the circle of Willis. These are by no means uncommon, and will be considered subsequently.

(*c*) Endarteritis and periarteritis in the cerebral vessels most commonly lead to apoplexy by the production of aneurisms, either miliary or coarse. There are instances in which the most careful search fails to reveal anything but diffuse degeneration of the cerebral vessels, particularly of the smaller branches; so that we must conclude that spontaneous rupture may occur without the previous formation of aneurism.

The hæmorrhage may be meningeal, cerebral, or intraventricular.

Meningeal Hæmorrhage may be outside the dura, between this membrane and the bone, or between the dura and arachnoid, or between the arachnoid and the pia mater. The following are the chief causes of this form of hæmorrhage: Fracture of the skull, in which case the blood usually comes from the lacerated meningeal vessels, sometimes from the torn sinuses. In these cases the blood is usually outside the dura or between it and the arachnoid. The next most frequent cause is rupture of aneurisms on the larger cerebral vessels. The blood is usually subarachnoid. An intracerebral hæmorrhage may burst into the meninges. A special form of meningeal hæmorrhage is found in the new-born, associated with injury during birth. And lastly, meningeal hæmorrhage may occur in the constitutional diseases and fevers. The blood may be in a large quantity at

the base; in cases of ruptured aneurism, particularly, it may extend into the cord or upon the cortex. Owing to the greater frequency of the aneurisms in the middle cerebral vessels, the Sylvian fissures are often distended with blood.

Intracerebral hæmorrhage is most frequent in the neighborhood of the corpus striatum, particularly toward the outer section of the lenticular nucleus. The hæmorrhage may be small and limited to the lenticular body and the internal capsule, or it may break the centrum ovale, or burst into the lateral ventricle, or extend to the insula. Hæmorrhages confined to the white matter—the centrum ovale—are rare. Localized bleeding may occur in the crura or in the pons. Hæmorrhage into the cerebellum is not uncommon, and usually comes from the superior cerebellar artery. The extravasation may be limited to the substance or rupture into the fourth ventricle. Twice I have known sudden death in girls under twenty-five to be due to cerebellar hæmorrhage.

Ventricular Hæmorrhage.—This rarely comes from the vessels of the plexuses or of the walls. It is not infrequent in early life and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year, and 14 under the twentieth year. In the cases which I have seen in adults it has almost always been caused by rupture of a hæmorrhage in the neighborhood of the caudate nucleus. The blood may be found in one ventricle only, but more commonly it is in both lateral ventricles, and may pass into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle, forming a complete mould in blood of the ventricular system.

Subsequent Changes.—The blood gradually changes in color, and ultimately the hæmoglobin is converted into the reddish-brown hæmatoidin. Inflammation occurs about the apoplectic area, limiting and confining it, and ultimately a definite wall may be produced, inclosing a cyst with fluid contents. In other instances a cyst is not formed, but the connective-tissue proliferates and leaves a pigmented scar. In meningeal hæmorrhage the effused blood may be gradually absorbed and leave only a staining of the membranes. In other cases, particularly in infants, when the effusion is cortical and abundant, there may be localized wasting of the convolutions and the production of a cyst in the meninges. Possibly certain of the cases of porencephaly are caused in this way.

Secondary degeneration follows when the motor cortex or motor path is involved. Thus, in persons dying some years after a cerebral apoplexy which has produced hemiplegia, the degeneration may be traced in the crus, in the anterior part of the pons, in the pyramidal fibres of the medulla, in the direct fibres of the cord of the same side, and in the crossed pyramidal fibres of the opposite side (Fig. 3).

Symptoms.—These may be divided into primary, or those connected with the onset, and secondary, or those which develop later after the early manifestations have passed away.

vessel.

The chest movements on the paralysed side may be
restricted, in rare instances on the opposite side (

Primary Symptoms.—Premonitory indications are rare. As a rule, the patient is seized while in full health or about the performance of some every-day action, occasionally an action requiring strain or extra exertion. Now and then instances are found in which there are sensations of numbness or tingling or pains in the limbs, or even choreiform movements in the muscles of the opposite side, the so-called prehemiplegic chorea. The onset of the apoplexy, as cerebral hæmorrhage is usually called, varies greatly. There may be sudden loss of consciousness and complete relaxation of the extremities. In such instances the name *apoplectic stroke* is particularly appropriate. In other cases the onset is more gradual and the loss of consciousness may not occur for a few minutes after the patient has fallen, or after the paralysis of the limbs is manifest. In the apoplectic attack the condition is as follows: There is deep unconsciousness; the patient cannot be roused. The face is injected, sometimes cyanotic, or of an ashen-gray hue. The pupils vary; usually they are dilated and inactive. The respirations are slow, noisy, and accompanied with stertor. Sometimes the Cheyne-Stokes rhythm may be present. The pulse is usually full, slow, and of increased tension. The temperature may be normal, but is often found subnormal, and, as in a case reported by Bastian, may sink below 95°. In cases of basal hæmorrhage the temperature, on the other hand, may be high. The urine and fæces are usually passed involuntarily. Convulsions are not common. It may be difficult to decide whether the condition is apoplexy associated with hemiplegia or sudden coma from other causes. An indication of hemiplegia may be discovered in the difference in the tonus of the muscles on the two sides. If the arm or the leg is lifted, it drops "dead" on the affected side, while on the other it falls more slowly. Rigidity also may be present. In watching the movements of the facial muscles in the stertorous respiration it will be seen that on the paralyzed side the relaxation permits the cheek to be blown out in a more marked manner. The head and eyes may be turned strongly to one side—conjugate deviation.

In other cases, in which the onset is not so abrupt, the patient may not lose consciousness, but in the course of a few hours there is loss of power, unconsciousness gradually develops, and deepens into profound coma. This is sometimes termed ingravescent apoplexy. The attack may occur during sleep. The patient may be found unconscious, or wakes to find that the power is lost on one side. Small hæmorrhages may cause hemiplegia without loss of consciousness, more particularly when they are in the territory of the central arteries.

Usually within forty-eight hours after the onset of an attack there is febrile reaction, and more or less constitutional disturbance associated with inflammatory changes about the hæmorrhage. The patient may die in this reaction, or, if consciousness has been regained, there may be delirium or recurrence of the coma. At this period the so-called early rigidity may develop in the paralyzed limbs. Trophic changes may occur,

such as sloughing or the formation of vesicles. The most serious of these is the sloughing eschar of the lower part of the back, or on the paralyzed side, which may appear within forty-eight hours of the onset and is usually of grave significance. The congestion at the bases of the lungs so common in apoplexy is regarded by some as a trophic change.

Conjugate Deviation.—In a right hemiplegia the eyes and head may be turned to the left side; that is to say, the eyes look toward the cerebral lesion. This is almost the rule in conjugate deviation of the head and eyes which occurs early in hemiplegia. When, however, convulsions or spasm develop or the state of so-called early rigidity in hemiplegia, the conjugate deviation of the head and eyes may be in the opposite direction; that is to say, the eyes look away from the lesion and the head is rotated toward the convulsed side. This symptom may be associated with cortical lesions, particularly, according to some authors, when in the neighborhood of the supramarginal and angular gyri. It may also occur in a lesion of the internal capsule or in the pons, but in the latter situation the conjugate deviation is the reverse of that which occurs in other cases, as the patient looks away from the lesion, and in spasm or convulsion looks toward the lesion. In cases in which consciousness is restored and the patient improves, the unilateral paralysis which persists is known as

Hemiplegia.—Hemiplegia is complete when it involves face, arm, and leg, or partial when it involves only one or other of these parts. This may be the result of a lesion (*a*) of the motor cortex; (*b*) of the pyramidal fibres in corona radiata and in the internal capsule; (*c*) of a lesion in the crus cerebri; or (*d*) in the pons Varolii (see Fig. 3, *x*, *y*, *z*). Hæmorrhage is perhaps the most common cause, but tumors and spots of softening may also induce it. The special details of the hemiplegia may here be considered. The face is involved on the same side as the arm and leg. This results from the fact that the facial muscles stand in precisely the same relation to the cortical centres as those of the arm and leg, the fibres of the upper motor segment of the facial nerve from the cortex decussating just as do those of the nerves of the limbs. The facial paralysis is partial, involving only the lower portion of the nerve, so that the orbicularis oculi and the frontalis muscles are uninvolved. The signs of the facial paralysis are usually well marked. There may be a slight difficulty in elevating the eyebrows or in closing the eye on the paralyzed side. The hypoglossal nerve also is involved. In consquence, the patient cannot put out the tongue straight, but it deviates toward the paralyzed side, inasmuch as the genio-hyo-glossus of the sound side is unopposed. When the hemiplegia is on the right side there may be aphasia.

The arm is, as a rule, more completely paralyzed than the leg. The loss of power may be absolute or partial. In severe cases it is at first complete. In others, when the paralysis in the face and arm is complete that of the leg is only partial. The face and arm may alone be par-

the

Or in rare cases the facial paralysis is complete but the movements may be present with involuntary as laughing or crying. (Care & Brevors N H Pat & Ep. 9.12.94)

Hemiwork in the cross, ure - pence in the cross.

Crossed hemiplegia. (a) cruc. (refer.

(b) non vul.

at first.

alyzed, while the leg escapes. Less commonly the leg is more affected than the arm, and the face may be only slightly involved.

Certain muscles escape in hemiplegia, particularly those associated in symmetrical movements, as the thoracic and abdominal muscles, a fact which Broadbent explains by supposing that as the spinal nuclei controlling these movements on both sides constantly act together, they may, by means of this intimate connection, be stimulated by impulses coming from only one side of the brain.

Crossed hemiplegia occurs when a lesion is in the lower section of the pons Varolii (Fig. 3, *z*), in which the facial nerve is involved as it passes through the pons after it has left its nucleus; whereas, the motor fibres involved in the lesion are above the point of their decussation, so that facial paralysis occurs on the same side as the lesion, and paralysis of the arm and leg on the opposite side.

The sensory disturbances are variable. Hemianæsthesia may coexist with hemiplegia, but in many instances there is only slight numbness of sensation. When the hemianæsthesia is marked, it is usually the result of a lesion in the internal capsule. In C. L. Dana's study of sensory localization he found that anæsthesia of organic cortical origin was always limited or more pronounced in certain parts, as the face, arm, or leg, and was generally incomplete. Total anæsthesia was either of functional or subcortical origin. Marked anæsthesia was much more common in softening than in hæmorrhage. Complete hemianæsthesia is certainly rare in hæmorrhage.

Disturbance of the special senses is not common. Hemianopia may exist on the same side as the lesion, and there may be diminution in the acuteness of the senses of hearing, taste, and smell.

As a rule, there is no wasting of the paralyzed limbs. The deep reflexes are increased on the paralyzed side, and ankle clonus may be present. The plantar and other superficial reflexes are usually diminished. The sphincters are not affected.

The *course* of the disease depends upon the situation and extent of the lesion. If slight, the hemiplegia may disappear completely within a few days or a few weeks. In severe cases the rule is that the leg gradually recovers before the arm, and the muscles of the shoulder girdle and upper arm before those of the forearm and hand. The face may recover quickly.

Except in the very slight lesions, in which the hemiplegia is transient, changes take place which may be grouped as

Secondary Symptoms.—These correspond to the chronic stage. In a case in which little or no improvement takes place within eight or ten weeks, it will be found that the paralyzed limbs undergo certain changes. The leg, as a rule, recovers enough power to enable the patient to get about, although the foot is dragged. In both arm and leg the condition of *secondary contraction* or *late rigidity* comes on and is always most marked in the upper extremity. The arm becomes permanently flexed at

the elbow and resists all attempts at extension. The wrist is flexed upon the forearm and the fingers upon the hand. The position of the arm and hand is very characteristic. There is frequently, as the contractures develop, a great deal of pain. In the leg the contracture is rarely so extreme. The loss of power is most marked in the muscles of the foot, and to prevent the toes from dragging the knee in walking is much flexed, or more commonly the foot is swung round in a half-circle.

The reflexes are at this stage greatly increased. These contractures are permanent and incurable, and are associated with a secondary descending sclerosis of the motor path. There are instances, however, in which rigidity and contracture do not occur, but the arm remains flaccid, the leg having regained its power. This *hémiplégie flasque* of Bouchard is found most commonly in children. Among other secondary changes in late hemiplegia may be mentioned the following: Tremor of the affected limbs, post-paralytic chorea, the mobile spasm known as athetosis, arthropathies in the joints of the affected side, and muscular atrophy. Athetosis and post-hemiplegic chorea will be considered in the hemiplegia of children. A word may here be said upon the subject of muscular atrophy of cerebral origin.

As a rule, atrophy is not a marked feature in hemiplegia, but in some instances it does develop. It has been shown to be due in some cases to secondary alterations in the gray matter of the anterior horns, as in a case reported by Charcot. Recently, however, attention has been called by Quincke to the fact that atrophy may follow as a direct result of the cerebral lesion. In his case, atrophy of the arm followed the development of a glioma in the anterior central convolutions. The gray matter of the anterior horns was normal. This wasting of cerebral origin occurs most frequently in children.

Diagnosis.—There are three groups of cases which offer increasing difficulty in recognition.

(1) Cases in which the onset is gradual, a day or two elapsing before the paralysis is fully developed and consciousness completely lost, are readily recognized, though it may be difficult to determine whether the lesion is due to thrombosis or to hæmorrhage.

(2) In the sudden apoplectic stroke in which the patient rapidly loses consciousness, the difficulty in diagnosis may be still greater, particularly if the patient is in deep coma when first seen.

The first point to be decided is the existence of hemiplegia. This may be difficult, although, as a rule, even in deep coma the limbs on the paralyzed side are more flaccid and drop instantly when lifted; whereas, on the non-paralyzed side the muscles retain some degree of tonus. The reflexes may be increased on the affected side and there may be conjugate deviation of the head and eyes. Rigidity in the limbs of one side is in favor of a hemiplegic lesion. It is practically impossible in a majority of

these cases to say whether the lesion is due to hæmorrhage, embolism, or thrombosis.

(3) Large hæmorrhage into the ventricles or into the pons may produce sudden loss of consciousness with complete relaxation, so that the condition may simulate coma from uræmia, alcoholism, opium poisoning, or epilepsy. The previous history and the mode of onset may give valuable information. In epilepsy convulsions have preceded the coma; in alcoholism there is a history of constant drinking, while in opium poisoning the coma develops more gradually; but in many instances the difficulty is practically very great, and on more than one occasion I have seen mortifying post-mortem disclosures under these circumstances. In ventricular hæmorrhage the coma is sudden and develops rapidly. The hemiplegic symptoms may be transient, quickly giving place to complete relaxation. Convulsions occur in many cases, and may be the very symptom to lead astray—as in a case of ventricular hæmorrhage which occurred in a puerperal patient, in whom, naturally enough, the condition was thought to be uræmic. Rigidity is often present. In hæmorrhage into the pons convulsions are frequent. The pupils may be strongly contracted, conjugate deviation may occur, and the temperature is apt to rise rapidly. The contraction of the pupils in pontine hæmorrhage naturally suggests opium poisoning. The difference in temperature in the two conditions is a valuable diagnostic point.

It may be impossible at first to give a definite diagnosis. In admissions to hospitals or in emergency cases the physician should be particularly careful about the following points: The examination of the head for injury or fracture; the urine should be tested for albumen and examined for sugar; a careful examination should be made of the limbs with reference to their degree of relaxation or the presence of rigidity, and the condition of the reflexes; the state of the pupils should be noted and the temperature taken. The most serious mistakes are made in the case of patients who are drunk at the time of the attack, a combination by no means uncommon in the class of patients admitted to hospital. Under these circumstances the case may be looked upon as one of alcoholic coma. It is best to regard each case as serious and to bear in mind that this is a condition in which, above all others, mistakes are common.

Prognosis.—From cortical hæmorrhage, unless very extensive, the recovery may be complete without a trace of contracture. This is more common when the hæmorrhage follows injury than when it results from disease of the arteries. Infantile meningeal hæmorrhage, on the other hand, is a condition which may produce idiocy or spastic diplegia.

Large hæmorrhages into the corona radiata and those which rupture into the ventricles rapidly prove fatal.

The hemiplegia which follows lesions of the internal capsule, the result of rupture of the artery of the corpus striatum, is usually persistent and followed by contracture. When the posterior fibres are involved

there may be hemianæsthesia, and later hemichorea or athetosis. In any case of cerebral apoplexy the following symptoms are of grave omen: persistence or deepening of the coma during the second and third day; rapid rise in temperature within the first forty-eight hours after the initial fall. In the reaction which takes place on the second or third day, the temperature usually rises, and its gradual fall on the third or fourth day with return of consciousness is a favorable indication. The rapid formation of bed-sores, particularly the malignant decubitus of Charcot, is a fatal indication. The occurrence of albumen and sugar, if abundant, in the urine is an unfavorable symptom.

When consciousness returns and the patient is improving, the question is anxiously asked as to the paralysis. The extent of this cannot be determined for some weeks. With slight lesions it may pass off entirely. If persistent at the end of a month some grade of permanent palsy is certain to remain, and gradually the late rigidity supervenes.

Embolism and Thrombosis (*Cerebral Softening*).

(*a*) **Embolism.**—The embolus usually enters the carotid, rarely the vertebral artery. In the great majority of cases it comes from the left heart and is either a vegetation of a fresh endocarditis or, more commonly, of a recurring endocarditis, or from the segments involved in an ulcerative process. Less often the embolus is a portion of a clot which has formed in the auricular appendix. Portions of clot from an aneurism, thrombi from atheroma of the aorta, or from the territory of the pulmonary veins, may also cause blocking of the branches of the circle of Willis. In the puerperal condition cerebral embolism is not infrequent. It may occur in women with heart-disease, but in other instances the heart is uninvolved, and the condition has been thought to be associated with the development of heart-clots, owing to increased coagulability of the blood. A majority of cases of embolism occur in ~~chronic~~ heart-disease. Cases are rare in the acute endocarditis of rheumatism, chorea, and febrile conditions. It is much more common in the secondary recurring endocarditis which attacks old sclerotic valves. The embolus most frequently passes to the left middle cerebral artery, as it enters the left carotid oftener than the right because of the more direct course of the blood in the former. The posterior cerebral and the vertebral are less often affected. A large plug may lodge at the bifurcation of the basilar. Embolism of the cerebellar vessels is rare.

Embolism occurs more frequently in women, owing, no doubt, to the greater frequency of mitral stenosis. Contrary to this general statement, Newton Pitt's statistics of 79 cases at Guy's Hospital indicate, however, that males are more frequently affected; for in this series there were 44 males and 35 females.

(*b*) **Thrombosis.**—Clotting of blood in the cerebral vessels occurs about

See Savelieu. Virchow archiv Jan 1894

8 9 per cent (Savelieu)

Savelieu gives 5·4 per cent in women

an embolus, as the result of a lesion of the arterial wall (either endarteritis with or without atheroma or, particularly, the syphilitic arteritis), in aneurisms both coarse and miliary, and very rarely as a result of abnormal conditions of the blood. Thrombosis of the cerebral vessels occasionally follows ligation of the carotid artery. The thrombosis is most common in the middle cerebral and in the basilar arteries.

Anatomical Changes following Thrombosis and Embolism.—Degeneration and softening of the territory supplied by the vessels is the ultimate result of the arterial obstruction. Blocking in a terminal artery may be followed by a condition resembling infarct, in which the territory is deeply infiltrated with blood. More commonly the change is much less striking, and the affected region may look only a little paler than normal or slightly softer. Gradually the process of softening proceeds, the tissue is infiltrated with serum and is moist, the nerve-fibres degenerate and become fatty. The neuroglia is swollen and œdematous. The color of the softened area depends upon the amount of blood. The hæmoglobin undergoes gradual transformation, and the early red color may give place to yellow. Formerly much stress was laid upon the difference between *red*, *yellow*, and *white* softening. The red and yellow are seen chiefly on the cortex. Sometimes the red softening is particularly marked in cases of embolism and in the neighborhood of tumors. The gray matter shows many punctiform hæmorrhages—capillary apoplexy. There is a variety of yellow softening—the *plaques jaunes*—common in elderly persons, which occurs in the gray matter of the convolutions. The spots are from one to two centimetres in diameter, the edges cleanly cut, and the softened area is represented by either a turbid, yellow material, or in some instances there is a space crossed by fine trabeculæ, in the meshes of which there is fluid. They result from fatty degeneration of the peripheral cortical arteries; less often the hyaline change is present. White softening occurs most frequently in the white matter, and is seen best about tumors and abscesses. Inflammatory changes are common in and about the softened areas. When the embolus is derived from an infected focus, as in ulcerative endocarditis, suppuration may follow. The final changes vary very much. The degenerated and dead tissue elements are gradually but slowly removed, and if the region is small may be replaced by growth of connective tissue and the formation of a scar. In larger regions the resorption results in the formation of a cyst, which may be crossed by connective-tissue trabeculæ. It is surprising for how long an area of softening may persist without much change.

The position and extent of the softening depend upon the obstructed artery. An embolus which blocks the middle cerebral at its origin involves both the arteries in the anterior perforated space and the cortical branches, and in such a case there is softening in the neighborhood of the corpus striatum, as well as in part of the region supplied by the cortical vessels. The freedom of anastomosis between these branches varies

a good deal. Thus, there are instances of embolism of the middle cerebral artery in which the softening has only involved the territory of the central branches, in which case blood has reached the cortex through the anterior and posterior cerebrals. When the middle cerebral is blocked (as is perhaps oftenest the case) beyond the point of origin of the central arteries, one or other of its branches is usually most involved. The embolus may lodge in the vessel passing to the third frontal convolution, or in the artery of the ascending frontal or ascending parietal; or it may lodge in the branch passing to the supramarginal and angular gyri, or it may enter the lowest branch which is distributed to the upper convolutions of the temporo-sphenoidal lobe. These are practically terminal arteries, and instances frequently occur of softening limited to a part, at any rate, of the territory supplied by them. Some of the most accurate focalizing lesions are in this way produced.

Symptoms.—Extensive thrombotic softening may exist without any symptoms. It is not uncommon in the post-mortem examination of the bodies of elderly persons to find the *plaques jaunes* scattered over the convolutions. So, too, softening may take place in the "silent" regions, as they are termed, without exciting any symptoms. When the central or cortical branches of the middle cerebral arteries are involved the symptoms are similar to those of hæmorrhage. Permanent or transient hemiplegia results. When the central arteries are involved the softening in the internal capsule is commonly followed by permanent hemiplegia. There are certain peculiarities associated with embolism and with thrombosis respectively.

In *embolism* the patient is usually the subject of heart-trouble, or there exist some of the conditions already mentioned. The onset is sudden, without premonitory symptoms. When the embolism blocks the left middle cerebral artery the hemiplegia is usually associated with aphasia. In *thrombosis*, on the other hand, the onset is more gradual; the patient has previously complained of headache, vertigo, tingling in the fingers; the speech may have been embarrassed for some days; the patient has had loss of memory or is incoherent, or paralysis begins at one part, as the hand, and extends slowly, and the hemiplegia may be incomplete or variable. Abrupt loss of consciousness is much less common, and when the lesion is small consciousness is retained. Thus, in thrombosis due to syphilitic disease, the hemiplegia may come on gradually without the slightest disturbance of consciousness.

The hemiplegia following thrombosis or embolism has practically the characteristics, both primary and secondary, described under hæmorrhage.

The following may be the effects of blocking the different vessels: (*a*) *Vertebral.*—The left branch is more frequently plugged. The effects are involvement of the nuclei in the medulla and symptoms of acute bulbar paralysis. It rarely occurs alone; more commonly with

(*b*) Blocking of the *basilar artery.* When this is entirely occluded,

there may be bilateral paralysis from involvement of both motor paths. Bulbar symptoms may be present; rigidity or spasm may occur. The temperature may rise rapidly. The symptoms, in fact, are those of apoplexy of the pons.

(*c*) The *posterior cerebral* supplies the occipital lobe on its inner face and the greater part of the temporo-sphenoidal lobe. Localized areas of softening may exist without symptoms. Blocking of the branch passing to the cuneus may be followed by hemianopia. Hemianæsthesia may result from involvement of the posterior part of the internal capsule.

(*d*) *Internal Carotid.*—The symptoms are variable. As is well known, the vessel is in a majority of cases ligated without risk. In other instances transient hemiplegia follows; in others again the hemiplegia is permanent. These variations depend on the anastomoses in the circle of Willis. If these are large and free, no paralysis follows, but in cases in which the posterior communicating and the anterior communicating vessels are small or absent, the paralysis may persist. In No. 7 of my Elwyn series of cases of infantile hemiplegia, the woman, aged twenty-four, when six years old, had the right carotid ligated for abscess following scarlet fever, with the result of permanent hemiplegia. Blocking of the internal carotid within the skull by thrombosis or embolism is followed by hemiplegia, coma, and usually death. The clot is rarely confined to the carotid itself, but spreads into its branches and may involve the ophthalmic artery.

(*e*) *Middle Cerebral.*—This is the vessel most commonly involved, and, as already mentioned, if plugged before the central arteries are given off, permanent hemiplegia usually follows from softening of the internal capsule. Blocking of the branches beyond this point may be followed by hemiplegia, which is more likely to be transient, involves chiefly the arm and face, and if on the left side is associated with aphasia. The individual branches passing to the third frontal, ascending parietal, to the supramarginal and angular gyri, or to the temporal gyri may be plugged.

(*f*) *Anterior Cerebral.*—No symptoms may follow, and even when the branches which supply the paracental lobule and the top of the ascending convolutions are plugged the branches from the middle cerebral are usually able to effect a collateral circulation in these parts. Hebetude and dulness of intellect may occur with obstruction of the vessel.

There is unquestionably greater freedom of communication in the cortical branches of the different arteries than is usually admitted, although it is not possible, for example, to inject the posterior cerebral through the middle cerebral, or the middle cerebral from the anterior; but the absence of softening in some instances in which smaller branches are blocked shows how completely may be the compensation. The dilatation of the collateral branches may take place very rapidly; thus a patient with chronic nephritis died about twenty-four hours after the hemiplegic attack. There were recent vegetations on the mitral and an embolus

in the right middle cerebral artery just beyond the first two branches (temporal). The central portion of the hemisphere was swollen and œdematous. The right anterior cerebral was greatly dilated, and by measurement its diameter was found to be nearly three times that of the left.

Treatment of Cerebral Hæmorrhage.—The patient should be placed with the head high, and measures immediately taken to reduce the arterial pressure. Of these the most rapid and satisfactory is venesection, which should be practiced whenever the arterial tension is much increased. With a small pulse of low tension and signs of cardiac weakness it is contra-indicated. The chief difficulty is in determining whether the apoplexy is really due to hæmorrhage, or to thrombosis or embolism, since in the latter group of cases bleeding probably does harm. As a rule, however, in middle-aged men with arterio-sclerosis, an accentuated aortic second sound, and hypertrophy of the left ventricle, bleeding is indicated. Horsley and Spencer have recently, on experimental grounds, recommended the practice, formerly employed empirically, of compression of the carotid, particularly in the ingravescent form; or even, in suitable cases, passing a ligature round the vessel. An ice-bag may be placed on the head and hot bottles to the feet. The bowels should be freely opened, either by calomel, or croton oil placed on the tongue. Counter-irritation to the neck or to the feet is not necessary. When dyspnœa, stertor, and signs of mechanical obstruction are present, the patient should be turned on the side, as recommended by Bowles. This procedure also lessens the liability to congestion of the lungs.

Special care should be taken to avoid bed-sores; and if bottles are used to the feet, they should not be too hot, since blisters may be readily caused by much lower temperature than in health. In the fever of reaction, aconite may be indicated, but should be cautiously used. Stimulants are not necessary, unless the pulse becomes feeble and signs of collapse supervene.

The treatment of *softening* from thrombosis or embolism is very unsatisfactory. Venesection is not indicated, as it lowers the tension and rather promotes clotting. If, as is often the case, the heart's action is feeble and irregular, stimulants and small doses of digitalis may be given with, if necessary, ether or ammonia. The bowels should be kept open, but it is not well to purge actively, as in hæmorrhage.

In the thrombosis which follows syphilitic disease of the arteries, and which is met with most frequently in men between twenty and forty (in whom the hemiplegia often sets in without loss of consciousness), the iodide of potassium should be freely used, giving from twenty to thirty grains three times a day, or, if necessary, larger doses. If the syphilis has been recent, mercurials are also indicated. Practically these are the only cases of hemiplegia in which we see satisfactory results from treatment.

Operative treatment has been suggested, and when the diagnosis of

subdural hæmorrhage can be made it is justifiable. An attempt to reach a central hæmorrhage in the neighborhood of the internal capsule would only increase the damage to the brain-substance. Very little can be done for the hemiplegia which remains. The damage is too often irreparable and permanent, and it is very improbable that iodide of potassium, or any other remedy, hastens in the slightest degree Nature's dealing with the blood-clot.

The paralyzed limbs may be gently rubbed once or twice a day, and this should be systematically carried out, in order to maintain the nutrition of the muscles and to prevent, if possible, contractures. After the lapse of a fortnight the muscles may be stimulated by the faradic current; but when contractures develop, electricity is useless, and the passive movements and frictions are alone indicated.

In a case of complete hemiplegia, the friends should at the outset be frankly told that the chances of full recovery are slight. Power is usually restored in the leg sufficient to enable the patient to get about, but in the majority of instances the finer movements of the hand are permanently lost. The general health should be looked after, the bowels regulated, and the secretions of the skin and kidneys kept active. In permanent hemiplegia in persons above the middle period of life, more or less mental weakness is apt to follow the attack, and the patient may become irritable and emotional.

And, lastly, when hemiplegia has persisted for more than three months and contractures have developed, it is the duty of the physician to explain to the patient, or to his friends, that the condition is past relief, that medicines and electricity will do no good, and that there is no possible hope of cure.

Aneurism of the Cerebral Arteries.

Miliary aneurisms are not included, but reference is made only to aneurism of the larger branches. The condition is not uncommon. There were twelve instances in my first eight hundred autopsies in Montreal.* This is a considerably larger proportion than in Newton Pitt's collection from Guy's Hospital, nineteen times in nine thousand inspections.

Etiology.—Males are more frequently affected than females. Of my twelve cases seven were males. The disease is most common at the middle period of life. One of my cases was a lad of six. Pitt describes one at the same age. The chief causes are (*a*) endarteritis, either simple or syphilitic, which leads to weakness of the wall and dilatation; and (*b*) embolism. As pointed out by Church, these aneurisms are often found with endocarditis. Pitt, in his recent study of the subject, concludes that it is exceptional to find cerebral aneurism unassociated with fungating

* Canada Medical and Surgical Journal, vol. xiv.

endocarditis. The embolus disappears, and dilatation follows the secondary inflammatory changes in the coats of the vessel.

Morbid Anatomy.—The middle cerebral branches are most frequently involved. In my twelve cases the distribution on the arteries was as follows: Internal carotid, 1; middle cerebral, 5; basilar, 3; anterior communicating, 3. With the exception of one case they were saccular and communicated with the lumen of the vessel by an orifice smaller than the circumference of the sac. In the 154 cases which make up the statistics of Lebert, Durand, and Bartholow the middle cerebral was involved in 44, the basilar in 41, internal carotid in 23, anterior cerebral in 14, posterior communicating in 8, anterior communicating in 8, vertebral in 7, posterior cerebral in 6, inferior cerebellar in 3 (Gowers). The size of the aneurism varies from that of a pea to that of a walnut. The hæmorrhage may be entirely meningeal with very slight laceration of the brain substance, but the bleeding may be, as Coats has shown, entirely within the substance.

Symptoms.—The aneurism may attain considerable size and cause no symptoms. In a majority of the cases the first intimation is the rupture and the fatal apoplexy. Distinct symptoms are most frequently caused by aneurism of the internal carotid, which may compress the optic nerve or the commissure, causing neuritis or paralysis of the third nerve. A murmur may be audible on auscultation of the skull. Aneurism in this situation may give rise to irritative and pressure symptoms at the base of the brain or to hemianopsia. In the remarkable case reported by Weir Mitchell and Dercum an aneurism compressed the chiasma and produced bilateral temporal hemianopsia.

Aneurism of the vertebral or of the basilar may involve the nerves from the fifth to the twelfth. A large sac at the termination of the basilar may compress the third nerves or the crura.

The diagnosis is, as a rule, impossible. The larger sacs produce the symptoms of tumor, and their rupture is usually fatal.

Endarteritis.

In no group of vessels do we more frequently see chronic degenerative changes than in those of the circle of Willis. The condition occurs as:

(*a*) *Arterio-sclerosis*, producing localized or diffused thickening of the intima with the formation of atheromatous patches or areas of calcification. In the later stages, as seen in elderly people, the arteries of the circle of Willis may be dilated, stiff, or almost universally calcified.

(*b*) *Syphilitic Endarteritis.*—As already mentioned under the section of syphilis, gummatous endarteritis is specially prone to attack the cerebral vessels. It has in itself no specific characters—that is to say, it is impossible in given sections to pick out an endarteritis syphilitica from

an ordinary endarteritis obliterans. On the other hand, as already stated, the nodular periarteritis is never seen except in syphilis.

Thrombosis of the Cerebral Sinuses and Veins.

The condition may be primary or secondary.

Primary thrombosis of the sinuses and veins is rare. It occurs (*a*) in children, particularly during the first six months of life, usually in connection with diarrhœa. It has, in my experience, been a rare condition. I have never seen an example of spontaneous thrombosis of the sinuses in a child, and only two instances, both in connection with meningitis, in which the cortical veins contained clots. Gowers believes that it is of frequent occurrence, and that thrombosis of the veins is not an uncommon cause of infantile hemiplegia.

(*b*) In connection with chlorosis and anæmia. Brayton Ball has recently called attention to this interesting association, and has reported one case and collected ten or eleven others from the literature. All were in girls with anæmia or chlorosis.

(*c*) In the terminal stages of cancer, phthisis, and other chronic diseases thrombosis may gradually occur in the sinuses and cortical veins. To the coagulum developing in these conditions the term marantic thrombus is applied.

Secondary Thrombosis is much more frequent and follows extension of inflammation from contiguous parts to the sinus wall. The common causes are disease of the internal ear, fracture, compression of the sinuses by tumor, or suppurative disease outside the skull, particularly erysipelas. In these cases the lateral sinus is most frequently involved. Of 57 fatal cases in which ear-disease caused death with cerebral lesions, there were 22 in which thrombosis existed in the lateral sinuses (Pitt). The thrombus may be small, or may fill the entire sinus and extend into the internal jugular vein. In more than one half of these instances the thrombus was suppurating. The disease spreads directly from the necrosis on the posterior wall of the tympanum. It is not so common in disease of the mastoid cells.

Symptoms.—Primary thrombosis of the longitudinal sinus may occur without exciting symptoms and is found accidentally at the post-mortem. There may be mental dulness with headache. Convulsions and vomiting may occur. In other instances there is nothing distinctive. In a patient who died under my care, at the Philadelphia Hospital, of phthisis, there was a gradual torpor, deepening to coma, without convulsions, localizing symptoms, or optic neuritis. The condition was thought to be due to a terminal meningitis. In the chlorosis cases the head symptoms have, as a rule, been marked. Ball's patient was dull and stupid, had vomiting, dilatation of the pupils, and double choked disks. Slight paresis of the left side occurred. An interesting feature in her case was

the development of swelling of the left leg. In the cases reported by Andrew, Church, Tuckwell, Isambard Owen, and Wilks the patients had headache, vomiting, and delirium. Paralysis was not present. In Douglas Powell's case, with similar symptoms, there was loss of power on the left side. Bristowe reports a case of great interest in an anæmic girl of nineteen, who had convulsions, drowsiness, and vomiting. Tenderness and swelling developed in the position of the right internal jugular vein, and a few days later on the opposite side. The diagnosis was rendered definite by the occurrence of phlebitis in the veins of the right leg. The patient recovered.

The onset of such symptoms as have been mentioned in an anæmic or chlorotic girl should lead to the suspicion of cerebral thrombosis. In infants the diagnosis can rarely be made. Involvement of the cavernous sinus may cause œdema about the eyelids or prominence of the eyes.

In the *secondary thrombi* the symptoms are commonly those of septicæmia. For instance, in over seventy per cent of Pitt's cases the mode of death was by pulmonary pyæmia. This author draws the following important conclusions: (1) The disease spreads oftener from the posterior wall of the middle ear than from the mastoid cells. (2) The otorrhœa is generally of some standing, but not always. (3) The onset is sudden, the chief symptoms being pyrexia, rigors, pains in the occipital region and in the neck, associated with a septicæmic condition. (4) Well-marked optic neuritis may be present. (5) The appearance of acute local pulmonary mischief or of distant suppuration is almost conclusive of thrombosis. (6) The average duration is about three weeks, and death is generally from pulmonary pyæmia. The chief points in the diagnosis may be gathered from these statements.

Pitt records an interesting case of recovery in a boy of ten, who had otorrhœa for years and was admitted with fever, earache, tenderness, and œdema. A week later he had a rigor, and optic neuritis developed on the right side. The mastoid was explored unsuccessfully. The fever and chills persisting, two days later the lateral sinus was explored. A mass of foul clot was removed and the jugular vein was tied, after which the boy made a satisfactory recovery.

III. AFFECTIONS OF THE SUBSTANCE.

I. Topical Diagnosis.

A majority of the lesions of the nervous system which permit of a local diagnosis have as an important part of their symptomatology disturbance of muscular action, and as our knowledge of the mechanism governing the movements of muscles is comparatively exact, we shall take this system as a basis for local diagnosis.

The motor system is made up of two segments, each consisting of groups of nerve-cells and their prolongations into nerve-fibres. The *upper segment* comprises the motor cortex and the pyramidal fibres; and the *lower segment* the motor cells in the medulla and cord and the nerve-fibres arising from them, forming the peripheral nerves distributed to the muscles, which may themselves be considered as part of this segment.

The nerve-cells are so arranged that when thrown into action, by whatever cause, a definite movement is the result, and the same combination of nerve-cells always causes the same movement, or, in other words, every movement of the body is represented in the nervous centres by combinations of the nerve-cells, or, as we say, is localized.

Movements are localised both in the cells of the lower segment and in those of the upper, and we have consequently spinal localization and cerebral localization.

Spinal Localization.—In the lower motor segment the muscles are represented in their simplest movements, and different sections of the cord have been found to represent the movements of different muscles. Our knowledge of this localization is by no means complete, but enough has been learned to aid us materially in determining the site of a spinal lesion.

The cells of the lower segment are found in the motor nuclei of the medulla, and in the anterior gray horns of the spinal cord. They are connected with the muscles by the axis cylinder processes, the anterior nerve-roots (roots of motor cranial nerves), the peripheral nerves, and the end organs by which they are brought into intimate relation with the protoplasm of the muscle fibre itself.

The following table prepared by Starr gives in detail our knowledge on this subject:

Localization of the Functions of the Segments of the Spinal Cord.

Segment	Muscles	Reflex	Sensation
II and III C.	Sterno-mastoid. Trapezius. Scalenus and rectus. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.

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Segment.	Muscles.	Reflex.	Sensation.
IV C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra and infra spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latisimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II to XII D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary regions causes retraction of epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.
I L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.

Segment.	Muscles.	Reflex.	Sensation.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Stroking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis antieus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I to II. S.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III to V. S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

Cerebral Motor Localization.—In the motor cortex the muscles are again represented, or, as Hughlings Jackson says, re-represented in their finer movements.

Motor Centres.—The experiments of Hitzig and Fritsch and of Ferrier, together with the previous clinical studies of Hughlings Jackson, laid the foundation of our present knowledge of cerebral localization.

The area for representation of the movements in the cerebral cortex is in the Rolandic region and comprises the ascending parietal and ascending frontal convolutions, the hinder part of the three frontal convolutions, and the parietal lobule, a continuation backward of the ascending parietal convolution (Fig. 2, motor region). This entire region is excitable, and stimulation by weak electrical currents produces muscular movements in the opposite half of the body. The centres presiding over the different groups of muscles may be thus classified:

(*a*) Centres for the trunk. These have been shown by Schäfer to be situated in the marginal gyrus, just within the longitudinal fissure, the region sometimes spoken of as the paracentral lobule.

(*b*) Centres for the lower limbs. These are situated at the upper part of the Rolandic region, close to the longitudinal fissure. As indicated in the diagram, the representation of movements of the different portions of the lower limb in this region is as follows (Fig. 9): Most anterior, the hip; next in order, the knee and ankle; then the big toe, the centre for

Segment.	Motor.	Reflex.	Sensory.
IV C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Serratus magnus. Rhomboid. Supra and infra spinatus.	Pupil. 4th to 7th cervical. Vital. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V C.	Deltoid. Biceps. Coraco-brachialis. Infra-spinatus. Serratus magnus. Supinator longus. Rhomboid. Teres minor. Extensors of wrist and fingers. Pronators.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces retraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI C.	Biceps. Brachialis anticus. Supinator longus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 8th cervical. Tapping elbow causes prolonged extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of fore-arm, front and back. Outer half of hand.
VII C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis costal part. Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and fore-arm. Radial half of the hand.
VIII C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I D.	Extensors of thumb. Intrinsic hand muscles. Flexor and hypothenar eminence.		Forearm front half, inner aspect of hand.
II or XII D.	Muscles of back and abdomen. Levator ani.	Epigastric. 4th to 9th dorsal. Tickling rectum or glans causes retraction of epigastrium. Abdominal. 7th to 12th dorsal. Stroking side of abdomen causes retraction of belly.	Site of shirt and abdomen to navel. Inner aspect of thigh.
I L.	Ilio-psoas. Sartorius. C. Muscles of abdomen.	Cremasteric. 1st to 4th lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and scrotum.

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IV L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
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I to II S.	Flexors of ankle. Long flexor of toes. Peronæi. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
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the movement of which surrounds the upper end of the fissure of Rolando. Still further back are the centres for movement of the small toes.

(*c*) Centres for the upper limbs. This area corresponds to about the

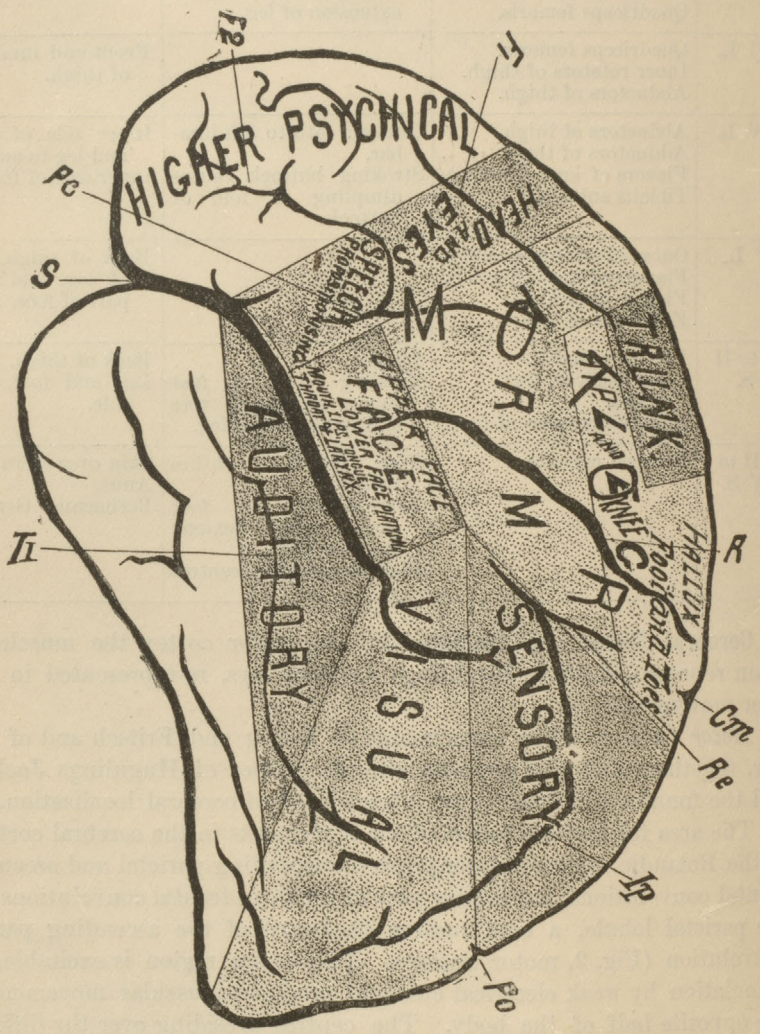


FIG. 2.—(After Mills). This diagram approximately indicates the views now held as a result of experiment and their confirmation or modification by clinico-pathological observation. It represents the division of the lateral surface of the cerebrum into higher psychical, motor, sensory, visual, and auditory areas; also the subdivision of the motor area into subareas for speech, the head and eyes, the face, arm, leg, and trunk. Only certain main points have been indicated by lettering, so as not to confuse: *S*, fissure of Sylvius; *R*, fissure of Rolando, or central fissure; *Pc*, precentral fissure; *Rc*, retrocentral fissure; *F1*, *F2*, superior and inferior frontal fissure; *Ip*, interparietal fissure; *Po*, parieto-occipital fissure; *T1*, first temporal fissure.

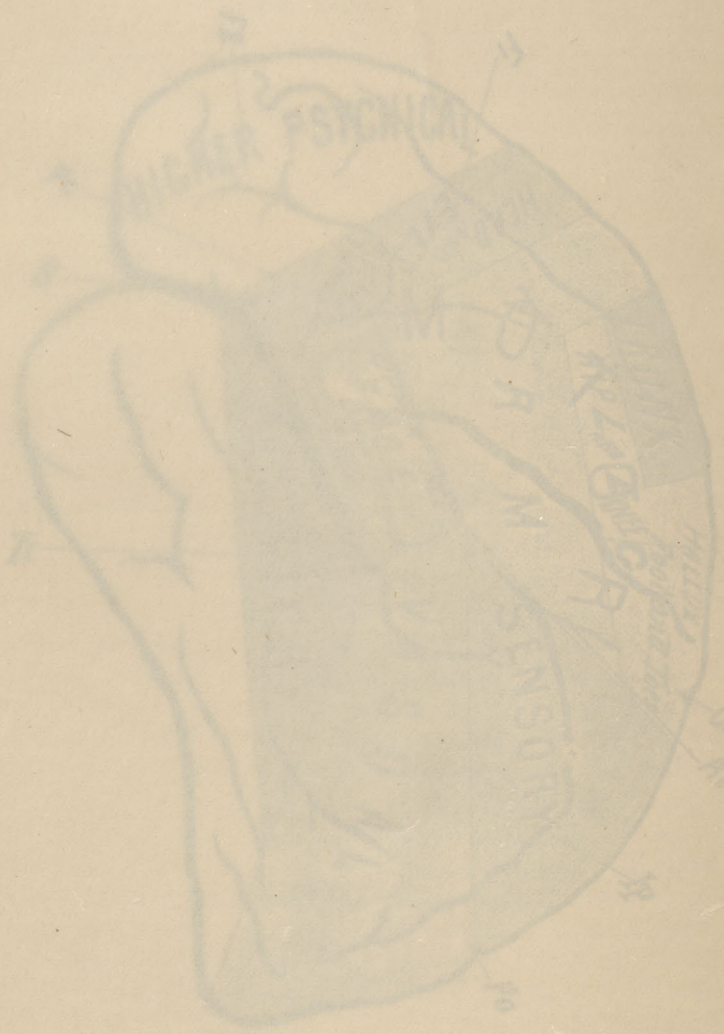
middle two fourths of the motor area. The careful studies of Horsley and Beevor have shown that from above downward the different segments of the limbs are represented as follows: Shoulder, elbow, wrist, fingers, the index-finger, and, lowest of all, the thumb.

(*d*) The centres for the face, tongue, pharynx, and larynx are situated in the lowest portion of the Rolandic area. The centres for the movement of the tongue and vocal cords are in the lower and anterior portion of the ascending convolution, and on the left side in man this region and the posterior part of the third left frontal convolution constitute the speech centre (Fig. 2), destruction of which is followed by one form of aphasia. In front of the precentral sulcus are centres for the representation of movements for turning the head and eyes to the opposite side.

The determination of these areas was worked out in animals and has now been thoroughly established in man, both by clinical observation and by the application of the electrodes in different situations during operations for the removal of growths in the brain or of the motor centres in epilepsy. The different regions must not be regarded as sharply separated from, but as blending with each other.

With these centres for voluntary movements are associated those which preside over the muscular sense, which is a compound of sensory impressions, of pressure, tension, and touch derived from the muscles as they are in motion. There is still dispute with reference to the localisation of this sense, but the general opinion is that lesions of the motor area itself cause slight loss both of muscular and tactile sense. Others place the centres for general sensation in the situation marked in Fig. 2.

The fibres uniting the cortical motor centres and the spinal centres have a long course, in which they probably have no connection with any other nerve-cells. They arise from the various centres, enter the white matter of the hemisphere (the corona radiata), and gradually converge to what is called the internal capsule, which lies between the lenticular nucleus and the thalamus and the caudate nucleus (Fig. 3). The position of the fibres in the internal capsule has been accurately worked out by several observers. The fibres from the centres of the face, tongue, eyes, and head occupy the most anterior position, just at the knee, as it is called, of the internal capsule, while the fibres from the upper extremities are just behind these, and those from the lower extremities occupy the position in the middle third of the posterior part. Leaving the internal capsule, the fibres forming the motor path pass from the brain into the crus, in which they occupy a lower and medial position. Passing through the pons, covered by the superficial layers of transverse fibres, they enter the medulla, of which they form the anterior or pyramidal tract. At the lower part of the medulla a large proportion of the fibres decussate and pass into the opposite side of the spinal cord, forming the crossed pyramidal tract of the lateral column, while a smaller number of the fibres descend in the anterior column of the same side, forming the direct pyramidal tract, or



middle two fourths of the motor area. The careful studies of Horsley and Beevor have shown that from above downward the different segments of the limbs are represented as follows: Shoulder, elbow, wrist, fingers, the index-finger, and, lowest of all, the thumb.

(*d*) The centres for the face, tongue, pharynx, and larynx are situated in the lowest portion of the Rolandic area. The centres for the movement of the tongue and vocal cords are in the lower and anterior portion of the ascending convolution, and on the left side in man this region and the posterior part of the third left frontal convolution constitute the speech centre (Fig. 2), destruction of which is followed by one form of aphasia. In front of the precentral sulcus are centres for the representation of movements for turning the head and eyes to the opposite side.

The determination of these areas was worked out in animals and has now been thoroughly established in man, both by clinical observation and by the application of the electrodes in different situations during operations for the removal of growths in the brain or of the motor centres in epilepsy. The different regions must not be regarded as sharply separated from, but as blending with each other.

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Türck's column. The pyramidal tracts diminish in size from above downward. The fibres enter the gray matter between the anterior and

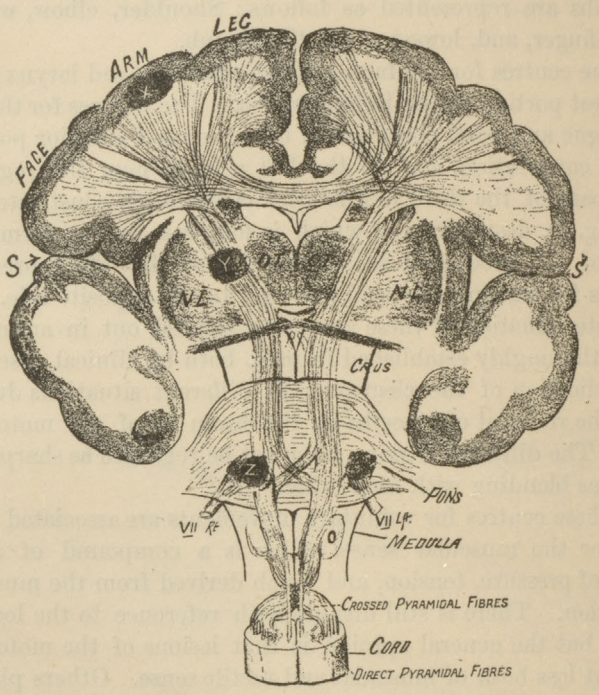


FIG. 3.—MOTOR TRACT (after Starr). *S*, fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *O*, olivary body. The tracts for the face, arm, and leg gather in the capsule and pass together to the lower pons, where the face-fibres cross to the opposite *VII* nerve nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral columns of the cord; the non-decussating fibres pass to the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure at *X*, *Y*, *Z*. At *Z* the lesion would involve the left facial nerve and the left pyramidal tract above the decussation, producing facial paralysis on the left side and paralysis of the arm and leg on the opposite side—crossed paralysis.

posterior cornua, pass forward, divide and subdivide, and finally join the plexus of the protoplasmic processes, and are in this way connected with the large nerve-cells of the anterior horns.

Lesions of the Motor System.—Each of the segments of the motor tract is to be considered as a nutritional unit, depending for its vitality upon the integrity of its ganglion cells. If certain cells in the cortex are destroyed, the fibres arising from them will degenerate throughout their length—that is, to the beginning of the lower motor segment. So also if the motor cells in the medulla or cord are injured, their nerve-fibres will degenerate, and the muscles to which they are distributed will also be involved in the process. The same thing occurs if the nerve-fibres become detached from their ganglion cells. This process is called secondary

degeneration or Wallerian degeneration, after the physician who first described it. Fig. 4 illustrates this process in the cortico-spinal motor segment.

The lesions may be grouped, as Hughlings Jackson suggested, into negative and positive, or, as they are now more usually termed, destructive and irritative.

Negative or destructive lesions anywhere in the motor path have as a result the abolishment of the functions of these parts—i. e., *paralysis*.

Positive or irritative lesions cause a perversion of the function—i. e., *abnormal muscular contractions*.

Although these two symptoms (paralysis and abnormal contractions) occur whenever the motor path is diseased, each of the segments imparts to them peculiar characteristics which enable us in a great majority of cases to determine the site of a lesion.

These characteristics depend upon, first, the special symptoms referable to the secondary degenerations in the two segments; second, upon their anatomical relation.

(*a*) **Lesions of the Lower or Spino-muscular Segment.** *Destructive Lesions.*—The destructive lesions cause here, as everywhere in the motor path, paralysis. We have seen above that when the nerve-fibres are cut off from their ganglion cells in the anterior horns, they not only degenerate themselves, but that the muscles to which they are distributed degenerate. This process is made evident by a change in the electrical reaction of the nerve and muscle—the reaction of degeneration—and the muscle becomes evidently atrophied. The myotatic irritability or muscle reflex, which depends upon the integrity of the lower motor segment, is lost in destructive lesions. This gives to the paralysis certain characteristics, namely, atrophy of the muscles, loss of its reflex excitability, and alteration of the electrical reactions of the nerve and muscle.

The anatomical relations of the lower motor segment also give certain peculiarities, which help to distinguish its lesions from those of the upper segment, on the one hand, and of the different parts of the lower segment on the other.

In general the different units which make up the lower segment are more or less widely separated from each other. An extreme example of

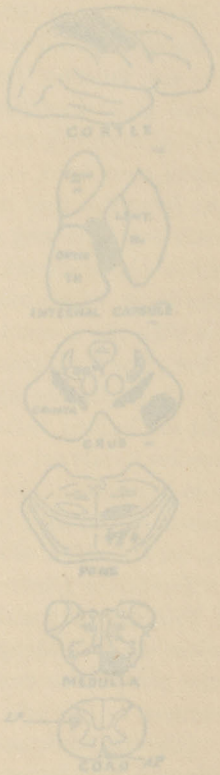
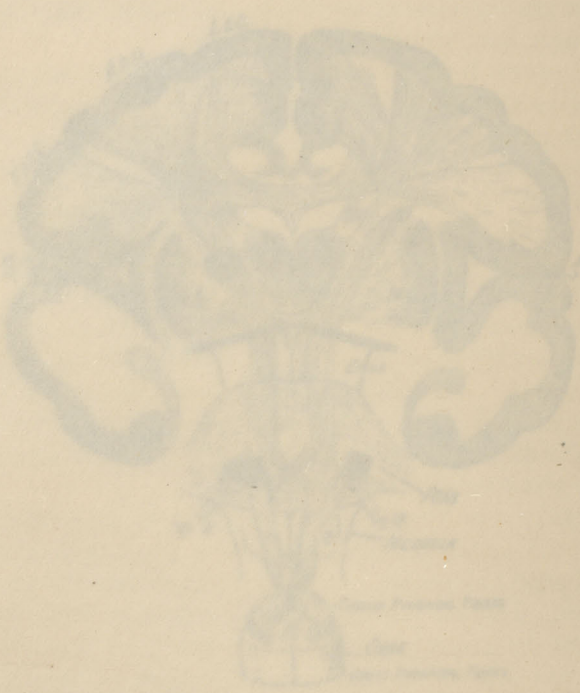


FIG. 4.—(After Gowers.) Diagram showing course and degeneration of pyramidal tract in right hemisphere, crus, pons medulla, and cord.



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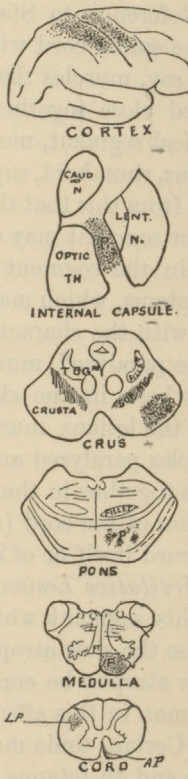

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this is the distance between the nucleus of the third nerve and the collection of motor cells in the lower part of the lumbar enlargement. For this reason lesions of this segment are more apt to cause paralysis of individual muscles or muscle groups, as distinguished from the more wide-spread paralysis due to lesions of the upper segment.

Reference to Starr's table will show that the muscles are represented in the spinal cord without relation to the nerves which supply them—that is to say, muscles that are supplied by a certain nerve may not be represented close together in the anterior horns; for instance, in the fourth cervical segment, movements of the diaphragm, deltoid, biceps, supinator longus, rhomboid, supraspinatus, and infraspinatus are represented. It follows from this that the distribution of a paralysis due to disease of the lower motor segment may enable us to distinguish the position of the lesion within the segment itself. We are often helped in this by the sensory symptoms, which may accompany the paralysis. Thus, if we have a paralysis with the characteristics of a lesion of the lower motor segment, and if the paralyzed muscles are all supplied by one nerve, and we discover anæsthesia in the skin of the arm supplied by that nerve, it is evident that the lesions must be in the nerve itself. On the other hand, if the muscles paralyzed are not supplied by a single nerve, but are represented close together in the spinal cord, and the anæsthesia corresponds to that section of the cord (see table), it is equally clear that the lesion must be of the cord itself or of its nerve-roots.

Irritative Lesions of the Lower Motor Segment.—We know of no lesion of this segment which has as its result abnormal muscular contraction unless the slow atrophy of the ganglion cells occurring in progressive muscular atrophy be considered as the cause of the fibrillary contraction so common in this affection.

(Certain tonic muscular contractions occurring in poisoning by strychnine and in tetanus are thought to be due to the perverted action of the lower motor centres, and Hughlings Jackson believes that certain convulsive paroxysms—"lowest level fits"—are due to discharging lesions of these centres, and claims laryngismus stridulus in this category.)

(*b*) **Lesions of the Upper, Cerebro-spinal Motor Segment.**—*Destructive lesions* cause, as in the lower motor segment, paralysis, and here again the secondary degeneration which follows the lesion gives to the paralysis its distinctive characteristics. In this case the paralysis is accompanied by a spastic condition, shown in an exaggeration of muscle reflex and an increase in the tension of the muscle. It is not accurately known how the degeneration of the pyramidal fibres causes this excess of the muscle reflex. The usual explanation is that under normal circumstances the upper motor centres are constantly exerting a restraining influence upon the activity of the lower centres, and that when the influence ceases to act, on account of disease of the pyramidal fibres, the latter take on increased activity, which is made manifest by an exaggeration of the muscle reflex.

after sensory symptoms — p. 9.

It was stated above that each segment of the motor path is to be considered as a nutritional unit and that the secondary degeneration in the upper segment stops at the beginning of the lower. So the muscles paralyzed by lesions in the upper segment do not undergo degenerative atrophy, nor do they present the reaction of degeneration.

The upper motor segment is much more compact than the lower, and for this reason a paralysis resulting from a lesion in it is apt to involve many muscles. This is especially true in regard to the pyramidal fibres, which run in a compact bundle, a lesion of which usually involves all of the fibres and causes a paralysis of all of the muscles of one side of the body—i. e., *hemiplegia.*

The motor centres of the cortex are more or less separated from each other, and a sharply localized lesion in this region causes a more limited paralysis, and cerebral monoplegias are the result; but even in this case the paralysis is diffuse, affecting the whole limb or a segment of the limb, and not individual muscles or groups of muscles.

To sum up, the paralyses due to lesions of the cerebro-spinal motor segment are diffuse, wide-spread, often hemiplegic; the paralyzed muscles are spastic (the tendon reflexes exaggerated), they do not undergo degenerative atrophy, and they do not present the degenerative reaction to electrical stimulation.

Irritative Lesions of the Upper Motor Segment.—Our knowledge of such lesions is confined for the most part to those acting on the cortical motor centres, and we know a number of processes which have as their result abnormal muscular contractions. These have as their type the localized convulsive seizures classed under Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex; for instance, such a convulsion beginning in the muscles of the face next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—i. e., they may destroy the nerve-cells of a certain centre, and either by their growth or presence may throw into abnormal activity those of the surrounding centres.

So far the motor system has been considered by itself, and we have endeavored to show how attention to the paralysis alone may help us to determine the seat of a lesion. It runs, however, in close connection with other systems of the nervous centre, which are often involved with it in morbid processes, giving rise to symptoms which aid us very much in making a local diagnosis.

Sensory Centres and Paths.—The association of the motor path with that for the conduction of sensory impressions is very intimate, but unfortunately our knowledge of the exact position of the sensory tracts is by

no means so precise. Some important facts are, however, known. Sensory fibres from different areas of the skin run in close connection with fibres of the lower motor segment in the mixed nerves. They separate from them and enter the spinal cord by the posterior roots. The regions which the different posterior roots supply is given in Starr's table. After entering the spinal cord the sensory fibres cross the middle line at once and pass up to the brain in the opposite half of the cord. Here they are again in close contact with the motor path, but with that of the other side of the body—i. e., the right half of the spinal cord contains the sensory fibres of the left side of the body and motor fibres of the right. The fibres which conduct the impressions for the muscular sense seem to be an exception and do not decussate in the cord. The exact position of the sensory paths in the cord is still somewhat uncertain, nor are we sure of their course in the medulla, pons, and peduncle. All the sensory fibres of the opposite side of the body are collected in the posterior third of the posterior limb of the internal capsule, just behind the motor fibres of the upper segment.

Much doubt and discussion still exist as to the areas for the representation of sensory impressions. Horsley has suggested that the muscular and tactile senses are localized in the motor cortex, and that two of the three chief layers of cells in this region subserve their functions. Dana's study shows that many lesions of the motor area, particularly in the hinder part, are associated with anæsthesia. On the other hand, Ferrier regards the hippocampal convolution, and Schäfer the gyrus fornicatus, as the centres for sensory impressions.

The centres for sight, hearing, smell, and taste have been referred to under the nerves ministering to these senses, and we shall consider the speech centres in the next section.

In the centrum ovale the fibres of the motor path are more or less closely associated with other systems of fibres; those connecting the cortex with nervous structures lying below it, projection fibres; the fibres which join the two hemispheres, commissural fibres; and those which join different parts of the same hemisphere, association fibres. Our knowledge of the function of these fibres leaves much to be desired.*

The following is a brief summary of the effects of lesions from the cortex to the spinal cord:

1. **The Cerebral Cortex.**—(*a*) Destructive lesions cause *spastic paralysis* in the muscles of the opposite side of the body. The extent of the paralysis depends upon that of the lesion. It is apt to be limited to the muscles of an extremity, giving rise to the cerebral monoplegias (Fig. 3, *X*). A lesion may involve two centres lying close together, thus producing paralysis of the face and arm, or of the arm and leg, but not of

* The student will find in Starr's work, Familiar Forms of Nervous Disease, an admirable presentation of this subject.

the face and leg without involvement of the arm. Very rarely the whole motor cortex is involved, causing paralysis of one side—cortical hemiplegia.

Combined with the muscular weakness there is usually some disturbance of sensation, particularly tactile impressions and those of the muscular sense.

(*b*) Irritative lesions cause localized spasms as described above. These convulsions are usually preceded and accompanied by sensory impressions. Tingling or pain, or a sense of motion in the part, is often the *signal symptom* (Seguin), and is of great importance in determining the seat of the lesion.

Lesions are often both destructive and irritative, and we have combinations of the symptoms produced by each. For instance, certain muscles may be paralyzed, and those represented near them in the cortex may be the seat of localized convulsions, or the paralyzed limb itself may be at times subject to convulsive spasms, or muscles which have been convulsed may become paralyzed. In this manner it is often possible to trace the progress of a lesion involving the motor cortex.

We have seen in a previous section that lesions involving the centres for the special senses may give rise to focal symptoms, and shall simply refer to them here. The symptoms caused by lesions of the speech centre will be described under aphasia, and it is only necessary to note the near situation of the motor speech area (Broca's centre) in the left third frontal convolution to the centres of the face and arm on that side, and to state that motor aphasia is often associated with monoplegia of the right side of the face and the right arm. Accompanying the paralysis following a Jacksonian fit of the right face or arm there is often a transient motor aphasia.

(2) **Centrum Ovale.**—Lesions in this part of the motor path cause paralysis, which has the distribution of a cortical palsy when the lesion is near the cortex, and of that due to a lesion of the internal capsule when it is near that region. They may be associated with symptoms due to the interruption of the other system of fibres running in the centrum ovale, and there may be sensory disturbances—hemianæsthesia and hemianopia—and if the lesion is in the left hemisphere one of the different forms of aphasia may accompany the paralysis.

(3) **Internal Capsule.**—Here all the fibres of the upper motor segment are gathered together in a compact bundle, and a lesion in this region is apt to cause complete hemiplegia of the opposite side, and if the lesion involves the hinder third of the posterior limb there is also hemianæsthesia, including even the special senses(Fig. 3, *Y*).

(4) **Crura Cerebri.**—Here, again, all the motor fibres and all the sensory fibres of the opposite side are collected in a small space, and a lesion may produce hemiplegia combined with sensory disturbances. On account of its anatomical relation the third cranial nerve is often involved

in lesions of the crus, causing paralysis of the muscles of the eye on the same side as the lesion combined with a hemiplegia of the opposite side—i. e., a crossed paralysis.

(5) **Pons.**—In the pons, medulla, and cord the upper and lower motor segments are both represented, the first by the pyramidal fibres, the latter by the motor nuclei and the nerve-fibres arising from them. Lesions here often affect both motor segments, and produce combinations of paralyses having the characteristics of each. Thus a lesion in the lower part of the pons may involve the pyramidal tract and cause a spastic paralysis of the opposite arm and leg, and also involve the nucleus or the fibres of the facial nerve, and so produce a paralysis of the same side of the face, accompanied by loss of the muscle reflex, atrophy, and the reaction of degeneration—crossed paralysis (Fig. 3, *Z*). The abducens and hypoglossus nerves may also be paralyzed in the same manner. In lesions of the pons the patient often has a tendency to fall toward the side on which the lesion is, due probably to implication of the middle peduncle of the cerebellum.

The symptoms produced by involvement of the different cranial nerves have been considered in detail in a previous section.

(6) **Spinal Cord.**—Unilateral lesions cause, first, a lower-segment paralysis, due to the disease of the centres at the site involved; second, a spastic paralysis of all the muscles on that side of the body below the lesion, due to interruption of the pyramidal fibres; and, third, disturbance of sensation in the opposite side of the body. (See under Brown-Séquard's paralysis.)

Transverse lesions of the cord cause paralysis with atrophy, etc., at the level of the lesion, spastic paralysis below it, combined with sensory disturbance and trouble with the bladder and rectum.

Affections of the peripheral nerves have already been considered.

II. Aphasia.

The speech mechanism consists of receptive, perceptive, and emissive centres in the cortex cerebri, disturbances of which cause *aphasia*, and centres in the medulla which preside over the muscles of articulation, disturbance of which produces *anarthria*, the condition of gradual loss of power of speech, such as occurs in bulbar paralysis.

The studies of Bastian, Küssmaul, Wernicke, Lichtheim, and others have widened enormously our knowledge of speech disorders. Language is gradually acquired by imitation. Thus, in teaching a child to say *bell*, the sound of the uttered word enters the afferent path (auditory nerve) and reaches the auditory perceptive centre, from which an impulse is sent to the emissive or motor centre presiding over the nuclei in the medulla, through which the muscles of articulation are set in action. The arc in Lichtheim's schema (Fig. 5) is a A, Mm. The child gradually ac-

Lesions of Cord localization in the Crura.

The crusta or foot of the cerebral peduncle contains exclusively white fibres, chiefly from the cortex. Descending degenerations have shown clearly the situation of some of these, of which the three groups (1) internal and deeper are fibres
does with lesions of the frontal lobe without motor paralysis. (2) the middle the larger, consisting of a small fasciculus which degenerates in affections of the foot of the ascending frontal convolution and contains the efferent fibres of the masticatory, infer. facial & hypoglossal nerves, the larger bundle consists of the motor pyramidal fasciculus.
(3) The most external fibres of the crusta are sensory and are connected with the occipital lobe. A few instances of degeneration have been noted Van Gudden No 1

The legmentum

The chief lesions other than secondary degenerations are as follows (1) Haemorrhage, which may arise from the vessels traversing the crus to reach the thalamus or, most important of all, rupture of the (motor ocular) artery of the middle In the classical case of Weber on section of the lower part of the left crus an oblong clot 15 mm in length, occupied the lay just below the internal & superior surface. The characteristic features are paralysis of arm face & leg of opposite side & motor ocular palsy of the same side, the crossed or alternate paralysis. The paralysis alterne pedunculaire of Millard supérieure of the French. The syndrome of Weber. Sensory and vaso motor changes have also been present. Haemorrhage into the legmentum is not necessarily associated with hemiplegia but there may be incomplete palsy of the 3rd & disturbance of sensation in the opposite side.

quires in this way *word memories*, which are stored at the centre A, and *motor memories*—the memories of the co-ordinated muscular movements necessary to utter words—which are stored at the centre M. In a similar manner, when shown the bell, the child acquires *visual memories*, which are conveyed through the optic nerve to the visual perceptive centres, o O. So also the memories of the sound of the bell when struck. The memory picture of the shape of the bell, the memory of the appearance of the word bell as written, and the motor memories of the muscular movements required to write the word are distinct from each other; yet they are intimately connected, and form together what is termed the *word-image*. In addition to all this the child gradually acquires in his education ideas as to the use of the bell—intellectual concepts—the centre for which is represented at I in the diagram In volitional or intellectual speech, as in uttering the word *bell*, the path would be I, M m, and in writing the word, I, M, W, h. These various "memories" are as a rule stored or centred in the left hemisphere.

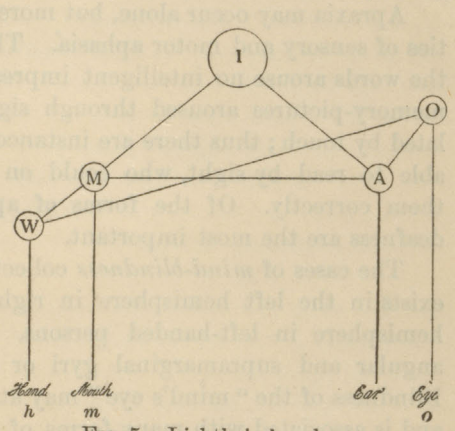


FIG. 5.—Lichtheim's schema.

The relations of written and spoken language are then with (*a*) sensory perceptive centres (hearing and sight and, in the blind, touch); (*b*) emissive or motor centres for speech and writing; and (*c*) psychical centres, through which we obtain an intellectual conception of what is said or written, and by which we express voluntarily our ideas in language.

There are two chief forms of aphasia—*sensory* and *motor*.

(1) **Sensory Aphasia; Apraxia; Word-blindness; Word-deafness.**—By apraxia is understood a condition in which there is loss or impairment of the power to recognize the nature and characteristics of objects. Persons so affected act "as if they no longer possessed such object memories, for they fail to recognize things formerly familiar. A fork, a cane, a pin, may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term blindness of mind was used, is found to extend to other senses than that of sight. Thus the tick of a watch, the sound of a bell, a melody of music, may fail to arouse the idea which it formerly awakened, and the patient has then deafness of mind, or an odor or taste no longer calls up the notion of the thing smelled or tasted; and thus it is found that each or all of the sensory organs, when

called into play, may fail to arouse an intelligent perception of the object exciting them. For the general symptoms of inability to recognize the use or import of an object the term *apraxia* is now employed." (Starr.)

Apraxia may occur alone, but more commonly is associated with varieties of sensory and motor aphasia. The patient may be able to read, but the words arouse no intelligent impression in his mind. While blind to memory-pictures aroused through sight, the perceptions may be stimulated by touch; thus there are instances on record of apraxic patients unable to read by sight, who could on tracing the letters by touch name them correctly. Of the forms of apraxia, mind-blindness and mind-deafness are the most important.

The cases of *mind-blindness* collected by Starr indicate that the lesion exists in the left hemisphere in right-handed persons, and in the right hemisphere in left-handed persons. The disease usually involves the angular and supramarginal gyri or the tracts proceeding from them. Blindness of the "mind's eye" may at times be functional and transitory, and is associated with many forms of mental disturbance. In a remarkable case reported by Macewen, the patient, after an injury to the head, had suffered with headache and melancholia, but there was no paralysis. He was psychically blind and though he could see everything perfectly well and could read letters, objects conveyed no intelligent impression. A man before his eyes was recognized as some object, but not as a man until the sounds of the voice led to the recognition through the auditory centres. The skull was trephined over the angular gyrus and the inner table was found to be depressed and a portion had been driven into the brain in this region. The patient recovered. Mind-blindness is the equivalent of visual amnesia.

Word-blindness may occur alone or with motor aphasia. In uncomplicated cases the patient is no longer able to recall the appearances of words, and does not recognize them on a printed or written page. The patient may be able to pronounce the letters and can often write correctly, but he cannot read understandingly what he has written. It is rare, however, for the patient to be able to write with any degree of facility. There are instances in which the patient, unable to read, has yet been able to do mathematical problems and to recognize play cards. The lesion in cases of word-blindness is, in a majority of cases, in the angular and supramarginal gyri on the left side. It is commonly associated with hemianopia, and not infrequently with mind-blindness.

Mind-deafness is a condition in which sounds, though heard and perceived as such, awaken no intelligent conceptions. A person who knows nothing of French has mind-deafness so far as the French language is concerned, and though he recognizes the words as words when spoken, and can repeat them, they awaken no auditory memories. The musical

faculties may be lost in aphasics, who may become note-deaf and unable to appreciate melodies or to read music. This may occur without the existence of motor aphasia, and on the other hand there are cases on record in which with motor aphasia for ordinary speech the patient could sing and follow tunes correctly. Mind-deafness is also known as auditory amnesia. Word-deafness is a condition in which the patient no longer understands spoken language. The memory of the sound of the word is lost, and can neither be recalled nor recognized when heard. It is usually associated with other varieties of aphasia, though there are cases in which the patient has been able to read and write and speak. The lesion in word-deafness has been accurately defined in a number of cases to be in the posterior portion of the first and second temporal convolutions on the left side (Fig. 2).

Other manifestations of mind-blindness are met with; thus a young man with secondary syphilis had several convulsive seizures, after one of which he remained unconscious for some time. On awakening, the memory-pictures of faces and places were a blank, and he neither knew his parents nor brothers, nor the streets of the town in which he lived. He had no aphasia proper, and no paralysis.

(2) **Motor or ataxic aphasia** is a condition in which the memory of the efforts necessary to pronounce words is lost, owing to disturbance in the emissive centres. This is the variety long ago recognized by Broca, the lesion of which was localized by him in the third left frontal convolution. In pure cases the patient is able to read (not aloud) and understands perfectly what is said. He may not be able to utter a single word; more commonly he can say one or two words, such as "no," "yes," and he not infrequently is able to repeat words. When shown an object, though not able to name it, he may evidently recognize what it is. If told the name, he may be able to repeat it. A man knowing the French and German languages may lose the power of expressing his thoughts in them, while retaining his mother-tongue; or, if completely aphasic, may recover one before the other. As the third left frontal convolution is in close contact with the centres for the face and arm, these are not uncommonly involved, with the production of a partial or, in some instances, a complete right-sided hemiplegia. *Alexia*, or inability to read, occurs with motor aphasia and also with word-blindness.

As a rule, in motor aphasia there is also inability to write—*agraphia*. When there is right brachial monoplegia it is difficult to test the capability, but there are instances of motor aphasia without paralysis, in which the power of voluntary writing is lost. The condition varies very much; thus a patient may not be able to write voluntarily or from dictation, and yet may copy perfectly. It is still a question whether there is a special writing centre. It has been placed by some writers at the base of the second frontal convolution, but in a

recent study Déjérine concludes that it is not separate from the speech centre.

There is a form known as *mixed aphasia*, or *paraphasia*, in which the patient understands what is said, and speaks even long sentences correctly, but he constantly tends to misplace words, and does not express his ideas in the proper words. All grades of this may be met with, from a state in which only a word or two is misplaced to an extreme condition in which the patient talks jargon. In these cases the association tract is interrupted between the auditory perceptive and the emissive centres, hence it is sometimes known as Wernicke's aphasia of conduction. The lesion is usually in the insula and in the convolutions which unite the frontal and temporal lobes. Lichtheim's schema will assist the student in obtaining a rational idea of the varieties of aphasia:

1. In the condition of apraxia or mind-blindness the ideation centres, I, are involved, often with the auditory and visual perceptive centres, A and O.

2. A lesion at A, the centre for the auditory memories of words (first left temporal gyrus), is associated with word-deafness.

3. A lesion at O, the centre for visual memories (angular and supramarginal gyri), causes word-blindness.

4. Interruption of the tracts uniting A M and O M causes the conduction aphasia of Wernicke—*paraphasia.*

5. Destruction of the centre M (Broca's convolution) causes pure motor aphasia, in which the patient cannot express thoughts in speech.

A lesion at M usually destroys also the power of writing, but, as stated, it is believed by many that the centre for writing, W, is distinct from that of speech. In this case a lesion at M, which would destroy the power of voluntary speech, might leave open the connections between O W and A W, by which the patient could copy or write from dictation.

The problems of aphasia are in reality excessively complicated, and the student must not for a moment suppose that cases are as simple as diagrams indicate. A majority of them are very complex, but with patience the diagnosis of the different varieties can often be worked out.

The following tests should be applied in each case of aphasia: (1) The power of recognizing the nature, uses, and relations of objects—i. e., whether apraxia is present or not; (2) the power to recall the name of familiar objects seen, smelled, or tasted, or of a sound when heard, or of an object touched; (3) the power to understand spoken words; (4) the capability of understanding printed or written language; (5) the power of appreciating and understanding musical tunes; (6) the power of voluntary speech—in this it is to be noted particularly whether he mis-

places words or not; (7) the power of reading aloud and of understanding what he reads; (8) the power to write voluntarily and of reading what he has written; (9) the power to copy; (10) the power to write at dictation; and (11) the power of repeating words.

Prognosis and Treatment.—In young persons the outlook is good, and the power of speech is gradually restored apparently by the education of the centres on the opposite side of the brain. In adults the condition is less hopeful, particularly in the cases of complete motor aphasia with right hemiplegia. The patient may remain speechless, though capable of understanding everything, and attempts at re-education may be futile. Partial recovery may occur, and the patient may be able to talk, but misplaces words. In sensory aphasia the condition may be only transient, and the different forms rarely persist alone without impairment of the powers of expression.

The education of an aphasic person requires the greatest care and patience, particularly if, as so often happens, he is emotional and irritable. It is best to begin by the use of detached letters, and advance, not too rapidly, to words of only one syllable. Children often make rapid progress, but in adults failure is only too frequent, even after the most pains-taking efforts. In the cases of right hemiplegia with aphasia the patient may be taught to write with the left hand.

III. Inflammation of the Brain

(Suppurative Encephalitis; Abscess).

Etiology.—Suppuration of the brain substance is rarely if ever primary, but results, as a rule, from extension of inflammation from neighboring parts or infection from a distance through the blood. The question of idiopathic brain abscess need scarcely be considered, though occasionally instances occur in which it is extremely difficult to assign a cause. There are three important etiological factors:

(1) Trauma. Falls upon the head or blows, with or without abrasion of the skin. More commonly it follows fracture or punctured wounds. In this group meningitis is frequently associated with the abscess.

(2) Extension of the inflammation from the neighboring parts, more particularly in caries of the petrous portion of the temporal bone, less frequently necrosis of the other bones, or extension of disease from the orbit. In this group otitis is the most important factor. There may be extension through the bone and involvement of the lateral sinus as already mentioned; but in other instances no direct connection can be traced and the infection is probably carried through the lymph channels.

(3) In septic processes. Abscess of the brain is not often found in pyæmia. In ulcerative endocarditis multiple foci of suppuration are

common. Localized bone-disease, suppuration in the liver, ~~but, above all,~~ certain ~~inflammations~~ of the lungs ~~(particularly gangrene, bronchiectasis, and fetid bronchitis),~~ are liable to be followed by abscess. It is an occasional complication of empyema. Abscess of the brain may follow the specific fevers. Bristowe has called attention to its occurrence as a sequel of influenza. The largest number of cases occur between the twentieth and fortieth years, and the condition is more frequent in men than in women.

Morbid Anatomy.—The abscess may be solitary or multiple, diffuse or circumscribed. In the acute, rapidly fatal cases following injury the suppuration is not limited; but in long-standing cases the abscess is enclosed in a definite capsule, which may have a thickness of from two to five millimetres. The pus varies much in appearance, depending upon the age of the abscess. In early cases it may be mixed with reddish *débris* and softened brain matter, but in the solitary encapsulated abscess the pus is distinctive, having a greenish tint, an acid reaction, and a peculiar odor, sometimes like sulphuretted hydrogen. The brain substance surrounding the abscess is usually œdematous and infiltrated. The size varies from that of a walnut to that of a large orange. There are cases on record in which the cavity has occupied the greater portion of a hemisphere. Multiple abscesses are usually small. In four fifths of all cases the abscess is solitary. Suppuration occurs most frequently in the cerebrum, and the temporo-sphenoidal lobe is more often involved than other parts. The cerebellum is the next most common seat, particularly in connection with ear-disease.

Symptoms.—Following injury or operation the disease may run an *acute* course, with fever, headache, delirium, vomiting, and rigors. The symptoms are those of an acute meningo-encephalitis, and it may be very difficult to determine, unless there are localizing symptoms, whether there is really suppuration in the brain substance. In the cases following ear disease the symptoms may at first be those of meningeal irritation. There may be irritability, restlessness, severe headache, and aggravated earache. Other striking symptoms, particularly in the more prolonged cases, are drowsiness, slow cerebration, vomiting, and optic neuritis. In the chronic form of brain abscess which may follow injury, otorrhœa, or local lung trouble, there may be a latent period ranging from one or two weeks to several months, or even a year or more. In the "silent" regions, when the abscess becomes encapsulated there may be no symptoms whatever during the latent period. During all this time the patient may be under careful observation and no suspicion be aroused of the existence of suppuration. Then severe headache, vomiting, fever, set in, perhaps with a chill. An Arab was admitted to my wards at the University Hospital in a condition of profound anæmia, having been picked up by the police in the street, covered with blood. There was a small localized area of dulness in the third and fourth interspaces on the right side close to the

are occasional causes

particularly Bronchiectasis which, with or without cirrhosis
was present in 17 of 38 cases of these so called "pulmonary
cerebral abscesses" collected by R. Lichtheimer
Med Chron. March 1884

sternum, and although no tubercle bacilli were found, it was thought to be probably a localized tuberculosis. He recovered rapidly from the anæmia, and within three months was strong and well. A few days before his intended discharge he began to complain of headache, which became aggravated. He had vomiting, fever, and gradually increasing coma. A large, solitary encapsulated abscess was found in the parieto-occipital region of the left hemisphere, and in the middle lobe of the right lung a circumscribed cavity, probably bronchiectatic, surrounded by fibroid tissue and containing a very offensive pus. So, too, after a blow upon the head or a fracture the symptoms of the lesion may be transient, and months afterward cerebral symptoms of the most aggravated character may develop.

The localization of the lesion is often difficult. In or near the motor region there may be convulsions or paralysis, and it is to be remembered that an abscess in the temporo-sphenoidal lobe may compress the lower motor centres and produce paralysis of the arm and face and on the left side cause aphasia. A large abscess may exist in the frontal lobe without causing paralysis, but in these cases there is almost always some mental dulness. In the temporo-sphenoidal lobe, the common seat, there may be no focalizing symptoms. So also in the parieto-occipital region; though here early examination may lead to the detection of hemianopia. In abscess of the cerebellum vomiting is common. If the middle lobe is affected there may be staggering—cerebellar incoördination. Localizing symptoms in the pons and other parts are still more uncertain.

Diagnosis.—In the acute cases there is rarely any doubt. The history of injury followed by fever, marked cerebral symptoms, the development of optic neuritis and rigors, delirium, and perhaps paralysis, make the diagnosis certain. In chronic ear-disease, such cerebral symptoms as drowsiness and torpor, with irregular fever, supervening upon the cessation of a discharge should excite the suspicion of abscess. It is particularly in the chronic cases that difficulties arise. The symptoms resemble those of tumor of the brain; indeed, they are those of tumor plus fever. In a patient with a history of trauma or with localized lung or pleural trouble, who for weeks or months has had slight headache or dizziness, the onset of a rapid fever, intense headache, and vomiting point strongly to abscess.

It is not always easy to determine whether the meninges are involved with the abscess. Often in ear-disease the condition is that of meningo-encephalitis. I have already referred to a condition sometimes associated with ear-disease, which may simulate closely cerebral meningitis or even abscess. Indeed, Gowers states that not only may these general symptoms be produced by ear-disease, but even distinct optic neuritis.

Treatment.—A remarkable advance has been made of late years in dealing with these cases, owing to the impunity with which the brain can be explored. In ear-disease free discharge of the inflammatory products should be promoted and careful disinfection practised. The treatment of injuries and fractures comes within the scope of the surgeon. The acute

symptoms, such as fever, headache, and delirium, must be treated by rest, an ice-cap, and, if necessary, local depletion. In all cases, when a reasonable suspicion exists of the occurrence of abscess, the trephine should be applied and the brain explored. The cases following ear-disease, in which the suppuration is in the temporo-sphenoidal lobe or in the cerebellum, offer the most favorable chances of recovery. The localization can rarely be made accurately in these cases, and the operator must be guided more by general anatomical and pathological knowledge. In cases of injury the trephine should be applied over the seat of the blow or the fracture. In ear-disease the suppuration is most frequent in the temporo-sphenoidal or in the cerebellum, and the operation should be performed at the points most accessible to these regions.

IV. HEMIPLEGIA AND DIPLEGIA IN CHILDREN.

It is as yet hard to say, without fuller knowledge of the etiology of these common conditions, where they should be classified. In a majority of the cases, whatever the nature of the primary pathological change, the final state is one of a chronic encephalitis, often with great atrophy of the convolutions or the formation of large cyst-like spaces—porencephalus.

I. Hemiplegia.

Etiology.—Of 135 cases, comprising those from the Infirmary for Diseases of the Nervous System, Philadelphia, from the Elwyn Institution for Feeble-minded Children, under Kerlin, and from my clinic at the Johns Hopkins Hospital, 60 were in boys and 75 in girls. Right hemiplegia occurred in 79, left in 56. In 15 cases the condition was said to be congenital.

In a great majority the disease sets in during the first or second year; thus of the total number of cases, 95 were under two. Cases above the fifth year are rare, only 10 in my series. Neither alcoholism nor syphilis in the parents appears to play an important *rôle* in this affection. Difficult or abnormal labor is responsible for certain of the cases, particularly injury with the *forceps*. Trauma, such as falls or puncturing wounds, is more rare. The condition followed ligation of the common carotid in one case.

Infectious diseases. All the authors lay special stress upon this factor. In 19 cases in my series the disease came on during or just after one of the specific fevers. I saw one case in which during the height of vaccination convulsions developed, followed by hemiplegia. In a great majority of the cases the disease sets in with a convulsion, in which the child may remain for several hours or longer, and after recovery the paralysis is noticed.

Morbid Anatomy.—In an analysis which I have made of 90 autopsies reported in the literature, the lesions may be grouped under three headings:

(*a*) Embolism, thrombosis, and hæmorrhage, comprising 16 cases, in 7 of which there was blocking of a Sylvian artery, and in 9 hæmorrhage. A striking feature in this group is the advanced age of onset. Ten of the cases occurred in children over six years old.

(*b*) Atrophy and sclerosis, comprising 50 cases. The wasting is either of groups of convolutions, an entire lobe, or the whole hemisphere. The meninges are usually closely adherent over the affected region, though sometimes they look normal. The convolutions are atrophied, firm, and hard, contrasting strongly with the normal gyri. The sclerosis may be diffuse and wide-spread over a hemisphere, or there may be nodular projections—the hypertrophic sclerosis. Some of the cases show remarkable unilateral atrophy of the hemisphere. In one of my cases the atrophied hemisphere weighed 169 grammes and the normal 653 grammes. The brain tissue may be a mere shell over a dilated ventricle.

(*c*) Porencephalus, which was present in 24 of the 90 autopsies. This term was applied by Heschel to a loss of substance in the form of cavities and cysts at the surface of the brain, either opening into and bounded by the arachnoid, and even passing deeply into the hemisphere, or reaching to the ventricle. In the study by Audrey of 103 cases of porencephalus, hemiplegia was mentioned in 68 cases.

Practically, then, in infantile hemiplegia cortical sclerosis and porencephalus are the important anatomical conditions. The primary change in the majority of these cases is still unknown. Porencephalia may result from a defect in development or from hæmorrhage at birth. The etiology is clear in the limited number of cases of hæmorrhage, embolism, and thrombosis, but there remains the large group in which the final change is sclerosis and atrophy. What is the primary lesion in these instances? The clinical history shows that in nearly all these cases the onset is sudden, with convulsions—often with slight fever. Strümpell believes that this condition is due to an inflammation of the gray matter—polio-encephalitis—a view which has not been very widely accepted, as the anatomical proofs are wanting. Gowers suggests that thrombosis may be present in some instances. This might probably account for the final condition of sclerosis, but clinically thrombosis of the veins rarely occurs in healthy children, which appear to be those most frequently attacked by infantile hemiplegia, and post-mortem proof is yet wanting of the association of thrombosis with the disease.

Symptoms.—(*a*) The onset. The disease may set in suddenly without spasms or loss of consciousness. In more than half the cases the child is attacked with partial or general convulsions and loss of consciousness, which may last from a few hours to many days. This is one of the most striking features in the disease. Fever is usually present. The

hemiplegia, noticed as the child recovers consciousness, is generally complete. Sometimes the paralysis is not complete at first, but develops after subsequent convulsions. The right side is more frequently affected than the left. The face is commonly not involved.

(*b*) Residual symptoms. In some cases the paralysis gradually disappears and leaves scarcely a trace as the child grows up. The leg, as a rule, recovers more rapidly and more fully than the arm, and the paralysis may be scarcely noticeable. In a majority of cases, however, there is a characteristic hemiplegic gait. The paralysis is most marked in the arm, which is usually wasted; the forearm is flexed at right angles, the hand is flexed, and the fingers are contracted. Motion may be almost completely lost; in other instances the arm can be lifted above the head. Late rigidity, which almost always develops, is the symptom which suggested the name *hemiplegia spastica cerebralis* to Heine, the orthopædic surgeon who first accurately described these cases. It is, however, not constant. The limbs may be quite relaxed even years after the onset. The reflexes are usually increased. In several instances, however, I have known them to be absent. Sensation is, as a rule, not disturbed.

Aphasia is a not uncommon symptom, and occurred in 16 cases of my series—a smaller number than given in the series of Wallenberg, Gaudard, and Sachs.

Mental Defects.—One of the most serious consequences of infantile hemiplegia is the failure of mental development. A considerable number of these cases drift into the institutions for feeble-minded children. Three grades may be distinguished—idiocy, which is most common when the hemiplegia has existed from birth; imbecility, which often increases with the development of epilepsy; and feeble-mindedness, a retarded rather than an arrested development.

Epilepsy.—Of the cases in my series, 41 were subjects of convulsive seizures, which is one of the most distressing sequences of the disease. The seizures may be either transient attacks of *petit mal*, true Jacksonian fits, beginning in and confined to the affected side, or general convulsions.

Post-hemiplegic Movements.—It was in cases of this sort that Weir Mitchell first described the post-hemiplegic movements. They are extremely common, and were present in 34 of my series. There may be either slight tremor in the affected muscles, or incoördinate choreiform movements—the so-called post-hemiplegic chorea—or, lastly,

Athetosis.—In this condition, described by Hammond, there are remarkable spasms of the paralyzed extremities, chiefly of the fingers and toes, and in rare instances of the muscles of the mouth. The movements are involuntary and somewhat rhythmical; in the hand, movements of adduction or abduction and of supination and pronation follow each other in orderly sequence. There may be hyperextension of the fingers, during which they are spread wide apart. This condition is much more frequent in children than in adults. In the latter it may be combined

with hemianæsthesia, and the lesion is not cortical, but basic in the neighborhood of the thalamus. The movements are sometimes increased by emotion. They usually persist during sleep.

II. Spastic Diplegia—Birth Palsies.

In this condition there is a paralysis with spasm of all extremities, dating from or shortly succeeding birth, more rarely following the fevers or an attack of convulsions. The legs are usually more involved than the arms; there is no wasting, no disturbance of sensation. The reflexes are increased. The mental condition is profoundly disturbed. The patients are usually imbeciles or idiots, helpless in mind and body. Ataxic and athetoid movements of the most exaggerated kind may occur.

While a limited number only of cases of infantile hemiplegia are congenital, on the other hand, in spastic diplegia a large proportion of the cases results from injury at birth. Practically the spastic paraplegia of children should be considered with this condition, as its etiology is essentially the same. The arms, too, may be so slightly affected as to make it difficult to determine whether it is a case of diplegia or paraplegia. The cases usually date from birth, and a majority are born in first labors or are forceps cases. Ross suggests that in feet presentation there may be laceration or tearing of the cerebro-spinal membranes.

Morbid Anatomy.—The birth palsies which ultimately induce the spastic diplegias or paraplegias are most frequently the result of meningeal hæmorrhage. The importance of this condition has been shown by the studies of Litzmann and Sarah J. McNutt. The bleeding may come from the veins, or, in one case which I saw with Hirst, from the longitudinal sinus. The bleeding has in many cases been thickest over the motor areas, and it seems probable that the sclerosis found in these cases may result from the compression of the blood-clot. In other instances the condition may be due to a fœtal meningo-encephalitis. In sixteen autopsies collected in the literature, in which the patients died at ages varying from two to thirty, the anatomical condition was either a diffuse atrophy, which was most common, or porencephalus.

Symptoms.—At first nothing abnormal may be noticed about the child. In some instances there have been early and frequent convulsions; then at the age when the child should begin to walk it is noticed that the limbs are not used readily, and on examination a stiffness of the legs and arms is found. Even at the age of two the child may not be able to sit up, and often the head is not well supported by the neck muscles. The rigidity, as a rule, is more marked in the legs, and there is adductor spasm. When supported on the feet, the child either rests on its toes and the inner surface of the feet, with the knees close together, or the legs may be crossed. The stiffness of the upper limbs varies. It may be scarcely noticeable or the rigidity may be as marked as in the legs. Con-

stant irregular movements of the arms are not uncommon. The child has great difficulty in grasping an object. The spasm and weakness may be more evident on one side than the other. The mental condition is, as a rule, defective and convulsive seizures are common.

Associated with the spastic paraplegia are two allied conditions of considerable interest, characterized by spasm and disordered movements. A child with spastic diplegia may present, in an unusual degree, irregular movements of the muscles. In attempting to grasp an object the fingers may be thrown out in a stiff, spasmodic, irregular manner, or there may be constant irregular movements of the shoulders, arms, and hands, with slight incoördination of the head. Cases of this description have been described as *chorea spastica*, and they may be difficult to separate from multiple sclerosis and from Friedreich's ataxia.

A still more remarkable condition is that of *bilateral athetosis*, in which there is a combination of spasm more or less marked with the most extraordinary bizarre movements of the muscles. The condition, as a rule, dates from infancy. The patient may not be able to walk. The head is turned from side to side; there are continual irregular movements of the face muscles, and the mouth is drawn and greatly distorted. The extremities are more or less rigid, particularly in extension. On making the slightest attempt to move, often spontaneously, there are extraordinary movements of the arms and legs, particularly of the arms, somewhat like though much more exaggerated than athetosis. The patients are often unable to help themselves on account of these movements. The reflexes are increased. The mental condition is variable. The patient may be idiotic, but in two of the four cases which I have seen the patients were intelligent. Massalongo,* who has carefully studied this condition, describes three cases in one family. I have collected fifty-three cases from the literature, thirty-three of which occurred in males and twenty in females. There have been three autopsies. In Kurella's case there was pachymeningitis and bilateral lesions of the motor convolutions. Déjérine's patient had atrophy of the convolutions on both sides, while in my case the brain macroscopically presented no changes.

III. Spastic Paraplegia.

This condition, which is more fully described under the section upon the spinal cord, is in reality a cerebral affection, and may be due to conditions similar to those found in spastic diplegia. Indeed, it may at first be difficult to determine whether the arms are involved or not. The evidence of the cerebral origin of the affection is based upon the frequent coexistence of idiocy, imbecility, and nystagmus, and the occurrence of cases of spastic diplegia, in which the paraplegic symptoms are identical. All grades are met with, from pure spastic paraplegia with perfect use of the

* Dell' Atetosi Doppia, Collezione Italiana di Letture sulla Medicina, Series V, N. 3.

arms to the most extreme bilateral spasm. There have been, so far as I know, only two autopsies in this disease: the case of Förster, in which there was a moderate grade of general cortical sclerosis with slight dilatation of the ventricles, and the recent case of Sachs, in which there was a meningo-encephalitis with atrophy and descending degeneration of both lateral columns.

Treatment.—The possibility of injury to the brain in protracted labor and in forceps cases should be borne in mind by the practitioner. The former entails the greatest risk. In infantile hemiplegia the physician at the outset sees a case of ordinary convulsions, perhaps more protracted and severe than usual. These should be checked as rapidly as possible by the use of the bromides, the application of cold or heat, and a brisk purge. During convulsions chloroform may be administered with safety even to the youngest children. When the paralysis is established not much can be hoped from medicines. In only rare instances does the paralysis entirely disappear. The indications are to favor the natural tendency to improve by maintaining the general nutrition of the child, to lessen the rigidity and contractures by massage and passive motion, and if necessary to correct deformities by mechanical or surgical measures. Much may be done by careful manipulation and rubbing and the application of a proper apparatus. In children the aphasia usually disappears. The epilepsy is a distressing and obstinate symptom, for which a cure can rarely be anticipated. Prolonged periods of quiescence are, however, not uncommon. In the Jacksonian fits the bromides rarely do good, unless there is much irritability and excitement. Operative measures, which have been carried out in several cases, have not been successful. The liability to feeble-mindedness is the most serious outlook in the infantile cerebral palsies. In many cases the damage is irreparable, and idiocy and imbecility result. With patient training and with care many of the children reach a fair measure of intelligence and self-reliance.

V. SCLEROSIS OF THE BRAIN.

General Remarks.—The connective tissue of the central nervous system is of two kinds—one, the neuroglia, special and peculiar, derived from the ectoderm, with distinct morphological and chemical characters; the other, derived from the mesoderm, identical with the ordinary collagenous fibrous tissue of the body. Both play important parts in indurative processes in the brain and cord. A convenient division of the cerebro-spinal scleroses is into degenerative, inflammatory, and developmental forms.

The *degenerative scleroses* comprise the largest and most important subdivision, in which provisionally the following groups may be made: (*a*) The common secondary degeneration which follows when nerve-fibres

are cut off from their trophic centres; (*b*) toxic forms, among which may be placed the scleroses from lead and ergot, and, most important of all, the sclerosis of the posterior column, due in such a large proportion of cases to the virus of syphilis. Other unknown toxic agents may possibly induce degeneration of the nerve-fibres in certain tracts. The systemic paths in the cord differ apparently in their susceptibility and the posterior columns appear most prone to undergo this change; (*c*) the sclerosis associated with change in the smaller arteries and capillaries, which is met with as a senile process in the convolutions. In all probability some of the forms of insular sclerosis are due to primary alterations in the blood-vessels; but it is not yet settled whether the lesion in these cases is a primary degeneration of the nerve cells and fibres to which the sclerosis is secondary, or whether the essential factor is an alteration in nutrition caused by lesions of the capillaries and smaller arteries.

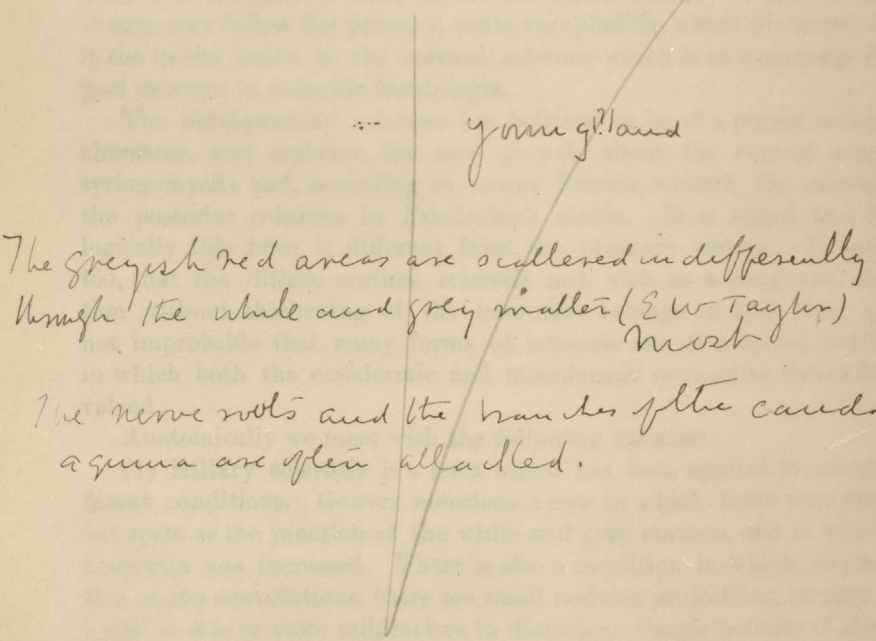
The *inflammatory scleroses* embrace a less important and less extensive group, comprising secondary forms which develop in consequence of irritative inflammation about tumors, foreign bodies, hæmorrhages, and abscess. Histologically these are chiefly mesodermic (vascular) scleroses, which arise from the connective tissue about the blood-vessels. Possibly a similar change may follow the primary, acute encephalitis, which Strümpell holds is the initial lesion in the cortical sclerosis which is so commonly found post mortem in infantile hemiplegia.

The *developmental scleroses* are believed to be of a purely neurogliar character, and embrace the new growth about the central canal in syringomyelia and, according to recent French writers, the sclerosis of the posterior columns in Friedreich's ataxia. It is stated that histologically this form is different from the ordinary variety. It may be, too, that the diffuse cortical sclerosis met with as a congenital condition without thickening of the meninges belongs to this type. It is not improbable that many forms of sclerosis are of a mixed character, in which both the ectodermic and mesodermic connective tissues are involved.

Anatomically we meet with the following varieties:

(1) **Miliary sclerosis** is a term which has been applied to several different conditions. Gowers mentions a case in which there were grayish-red spots at the junction of the white and gray matters, and in which the neuroglia was increased. There is also a condition in which, on the surface of the convolutions, there are small nodular projections, varying from a half to five or more millimetres in diameter. Single nodules of this sort are not uncommon; sometimes they are abundant. So far as is known no symptoms are produced by them.

(2) **Diffuse sclerosis,** which may involve an entire hemisphere, or a single lobe, in which case the term *sclérose lobaire* has been applied to it by the French. It is not an important condition in general medical practice, but occurs most frequently in idiots and imbeciles. In extensive


young gl.? and
The greyish red areas are scattered in different
through the white and grey smaller (E W Taylor)
most
The nerve roots and the trunks of the cauda
equina are often attacked.

cortical sclerosis of one hemisphere the ventricle is usually dilated.* The symptoms of this condition depend upon the region affected. There may be a considerable extent of sclerosis without symptoms or without much mental impairment. In a majority of cases there is hemiplegia or diplegia with imbecility or idiocy.

(3) **Tuberous Sclerosis.**—In this remarkable form, which is also known as hypertrophic, there are on the convolutions areas projecting beyond the surface of an opaque white color and exceedingly firm. The sclerosis may not disturb the symmetry of the convolution, but simply cause a great enlargement, increase in the density, and a change in the color.

These three forms are not of much practical interest except in asylum and institution work. The last variety forms a well-characterized disease of considerable importance, namely:

(4) Insular Sclerosis (*Sclérose en plaques*).

Definition.—A chronic affection of the brain and cord, characterized by localized areas in which the nerve elements are more or less replaced by connective tissue. This may occur in the brain or cord alone, more commonly in both.

Etiology.—This is obscure. Kahler, Marie, and others assign great importance to the infectious diseases, particularly scarlet fever. It is found most commonly in ~~middle-aged~~ persons, ~~but~~ cases are not uncommon in children, in whom Pritchard states that more than fifty cases have been reported.†

Morbid Anatomy.—The sclerotic areas are widely distributed through the brain and cord, ~~Cases limited to the cord~~ are almost unknown. ~~On section of the brain, grayish-red areas are seen scattered through the white matter. The cortex is not often involved.~~ The patches are ~~sometimes~~ abundant in the neighborhood of the ventricles, and in the pons, cerebellum, basal ganglia, and the medulla. The cord may be only slightly involved or there may be irregular areas in different regions. Histologically in the sclerosed patches there is great increase in the connective tissue, the fibres of which are denser and firmer. The gradual growth destroys the medulla of the nerves, but the axis cylinders persist in a remarkable way.

Symptoms.—The onset is slow and the disease is chronic. Feebleness of the legs with irregular pains and stiffness are among the early symptoms. Indeed, the clinical picture may be that of spastic paraplegia with great increase in the reflexes. The following are the most important features:

(*a*) *Volitional Tremor.*—There is no paralysis of the arms, but on at-

* In my monograph on Cerebral Palsies of Children I have given a description of the distribution of the sclerosis in ten specimens in the museum at the Elwyn Institution.

† Cyclopedia of the Diseases of Children, vol. iv.

tempting to pick up an object there is trembling or rapid oscillation. A patient may be unable to lift even a glass of water to the mouth. The tremor may be marked in the legs and in the head, which shakes as he walks. When the patient is recumbent the muscles may be perfectly quiet. On attempting to raise the head from the pillow, trembling at once comes on. (*b*) *Scanning Speech.*—The words are pronounced slowly and separately, or the individual syllables may be accentuated. This staccato or syllabic utterance is a common feature. (*c*) Nystagmus, a rapid oscillatory movement of both eyes, constitutes an important symptom.

Sensation is unaffected in a majority of the cases. Optic atrophy sometimes occurs, but not so frequently as in tabes. The sphincters, as a rule, are unaffected until the last stages. Mental debility is not uncommon. Remarkable remissions occur in the course of the disease, in which for a time all the symptoms may improve. Vertigo is common, and there may be sudden attacks of coma, such as occur in general paresis.

The *diagnosis* in well-marked cases is easy. Volitional tremor, scanning speech, and nystagmus form a characteristic symptom-group. With this there is usually more or less spastic weakness of the legs. Paralysis agitans, certain cases of general paresis, and occasionally hysteria may simulate the disease very closely. If the case is not seen until near the end the diagnosis may be impossible. Buzzard holds that of all organic diseases of the nervous system disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria.

Much more puzzling, however, are the instances of *pseudo-sclérose en plaques*, which have been described by Westphal. The volitional tremor, the scanning speech, and the spastic condition are present, but no lesions have been found post mortem. The movements in this form are more violent, but nystagmus does not occur. Some of the cases may possibly be examples of general paresis. In children the condition may with difficulty be separated from Friedreich's ataxia.

The *prognosis* is unfavorable. Ultimately, the patient, if not carried off by some intercurrent affection, becomes bedridden.

Treatment.—No known treatment has any influence on the progress of sclerosis of the brain. Neither the iodides nor mercury have the slightest effect, but a prolonged course of nitrate of silver may be tried.

VI. CHRONIC DIFFUSE MENINGO-ENCEPHALITIS

(*Dementia Paralytica; General Paresis*).

Definition.—A chronic, progressive meningo-encephalitis associated with psychical and motor disturbances, finally leading to dementia and paralysis.

Etiology.—Males are affected much more frequently than females. It occurs chiefly between the ages of thirty and fifty-five. Heredity is a

9. 3. 94 . I saw today with Beevor at the N H for Paralysed & Epileptic a man, aged about 28 who about a year ago after burying a child began to have trouble first in one leg & then in the other, weakness & stiffness which have continued & gradually worse. but he has kept at work.

now. has. (1) extreme spastic paraplegia, gait most characteristic. no wasting. (2) no Romberg symptom. (3) no ocular symptoms. except. Nystagmus. on forced lateral movements of the eyes & fixing them an important point in testing slight grades of hyperkinesia. wrist-jerk. & jaw jerk. (5) slight tremor on outstretched extension. touches tip of nose well but with slight incoordination. (6) no scanning speech.

Beevor diagn. insular sclerosis Remarkable that so advanced a case (condition) of spastic paraplegia should have so few cerebral signs – stiff. spinal cord.

the points to be relied upon in the differential are in order of importance, the nystagmus, the bladder disturbances & the outstretched tremor. The tremor in hysteria is not rotational

French writers regard these cases as instances of hysterical tremor (see Lloyd. Am Jr Med S. 1893 ii

and arsenic is recommended

An excellent resumé by Klippel on the Spinal symptoms a spinal form of paresis. A. Exp. Path. Jan 1894

factor in only a few cases. An overwhelming majority of the cases are in married people. Statistics show that it is more common in the lower classes of society, but in this country in general medical practice the disease is certainly more common in the well-to-do classes. An important predisposing cause is "a life absorbed in ambitious projects with all its strongest mental efforts, its long-sustained anxieties, deferred hopes, and straining expectation" (Mickle). The habits of life so frequently seen in active business men in our large cities, and well expressed by the phrase "burning the candle at both ends," strongly predispose to the disease. Among other factors of importance are syphilis, excesses *in baccho et venere*, injuries, and chronic lead-poisoning.

Morbid Anatomy.—The essential histological changes in the cerebral cortex are thus summarized by Bevan Lewis: (1) A stage of inflammatory change in the tunica adventitia of the arteries with excessive nuclear proliferation, profound changes in the vascular channels, and trophic changes induced in the tissues around.

(2) A stage of extraordinary development of the lymph-connective system of the brain, with a parallel degeneration and disappearance of nerve elements and the axis cylinders of which they are denuded.

(3) A stage of general fibrillation with shrinking and extreme atrophy of the parts involved.

The macroscopical changes are: (*a*) Increase in the cerebro-spinal fluid, œdema of the pia, and thickening and opacity of the meninges, which are adherent in places and tear the cortex on removal. The dura is sometimes thickened, and pachymeningitis hæmorrhagica interna may be present.

(*b*) The convolutions are atrophied, usually in a marked degree, and in consequence the brain looks small. This is particularly noticeable in the frontal and parietal regions. On section it cuts with firmness. In extreme cases the gray matter may be obscurely outlined. The grade of sclerosis varies much in different cases. The white matter may be firmer in consistence, but it does not show such important changes. The ventricles are dilated and the ependyma extremely granular. In addition, there are frequently areas of softening or hæmorrhage associated with chronic arterio-sclerosis.

(*c*) Spinal cord. Changes occur leading to increase in the connective-tissue elements and frequently to degeneration of the pyramidal tracts secondary to the cortical lesion. The posterior columns may also be involved, and occasionally the distribution of the sclerosis is that of the amyotrophic form.

Symptoms.—(*a*) *Prodromal Stage.*—This is of variable duration, and is characterized by a general mental state which finds expression in symptoms trivial in themselves but important in connection with others. Irritability, inattention to business amounting sometimes to indifference or apathy, and sometimes a change in character marked by acts which

may astonish the friends and relatives may be the first indications. Instead of apathy or indifference there may be an extraordinary degree of physical and mental restlessness. The patient is continually planning and scheming, or may launch into extravagances and speculation of the wildest character. A common feature at this period is the display of an unbounded egoism. He boasts of his personal attainments, his property, his position in life, or of his wife and children. Following these features are important indications of moral perversion, manifested in offences against decency or the law, many of which acts have about them a suspicious effrontery. Forgetfulness is common, and may be shown in inattention to business details and in the minor courtesies of life. At this period there may be no motor phenomena. The onset of the disease is usually insidious, although cases are reported in which epileptiform or apoplectiform seizures were the first symptoms. Among the early motor features are tremor of the tongue and lips in speaking, slowness of speech and hesitancy, and inequality of the pupils.

(*b*) *Second Stage.*—This is characterized in brief by mental exaltation or excitement and a progress in the motor symptoms. "The intensity of the excitement is often extreme, acute maniacal states are frequent; incessant restlessness, obstinate sleeplessness, noisy, boisterous excitement, and blind, uncalculating violence especially characterize such states" (Lewis). It is at this stage that the delusion of grandeur becomes marked and the patient believes himself to be possessed of countless millions or to have reached the most exalted sphere possible in profession or occupation. This expansive delirium, as it is called, is, however, not characteristic, as was formerly supposed, of paralytic dementia. Besides, it does not always occur, but in its stead there may be marked melancholia or hypochondriasis, or, in other instances, alternate attacks of delirium and depression.

The facies has a peculiar stolidity, and in speaking there is marked tremulousness of the lips and facial muscles. The tongue is also tremulous, and may be protruded with difficulty. The speech is slow, interrupted, and blurred. Writing becomes difficult on account of unsteadiness of the hand. The subject matter of the patient's letters give valuable indications of the mental condition. In many instances the pupils are unequal, irregular, sluggish, sometimes large. Important symptoms in this stage are apoplectiform seizures and paralysis. There may be slight syncopal attacks in which the patient turns pale and may fall. Some of these are *petit mal.* In the true apoplectiform seizure the patient falls suddenly, becomes unconscious, the limbs are relaxed, the face is flushed, the breathing stertorous, the temperature increased, and death may occur. The epileptic seizures are more common than the apoplectiform and may occur early in the disease. A definite aura is not uncommon. The attack usually begins on one side and may not spread. There may be twitchings either in the facial or brachial muscles. Typical Jack-

Absence of the pain reaction on pressure upon the ulnar nerve behind the elbow (Biernacik symptom) is apparently not of any special value.

sonian epilepsy may occur. In a case which died recently under my care, these seizures were among the early symptoms and the disease was regarded as cerebral syphilis. Paralysis, either monoplegic or hemiplegic, may follow these epileptic seizures, or may come on with great suddenness and be transient. In this stage the gait becomes impaired, the patient trips readily, has difficulty in going up or down stairs, and the walk may be spastic or occasionally tabetic. This paresis may be progressive. The knee-jerk is usually increased. Bladder or rectal symptoms gradually develop. The patient becomes helpless, bedridden, and completely demented, and unless care is taken may suffer from bedsores. Death occurs from exhaustion or from some intercurrent affection.

Diagnosis.—The recognition of the disease in the earliest stage is extremely difficult, as it is often impossible to decide that the slight alteration in conduct is anything more than one of the moods or phases to which most men are at times subject. The following description by Folsom is an admirable presentation of the diagnostic characters of the early stage of the disease: "It should arouse suspicion if, for instance, a strong, healthy man, in or near the prime of life, distinctly not of the 'nervous,' neurotic, or neurasthenic type, shows some loss of interest in his affairs or impaired faculty of attending to them; if he becomes varyingly absent-minded, heedless, indifferent, negligent, apathetic, inconsiderate, and, although able to follow his routine duties, his ability to take up new work is, no matter how little, diminished; if he can less well command mental attention and concentration, conception, perception, reflection, judgment; if there is an unwonted lack of initiative, and if exertion causes unwonted mental and physical fatigue; if the emotions are intensified and easily change, or are excited readily from trifling causes; if the sexual instinct is not reasonably controlled; if the finer feelings are even slightly blunted; if the person in question regards with a placid apathy his own acts of indifference and irritability and their consequences, and especially if at times he sees himself in his true light and suddenly fails again to do so; if any symptoms of cerebral vaso-motor disturbances are noticed, however vague or variable."

There are cases of cerebral syphilis which closely simulate dementia paralytica. The mode of onset is important, particularly since paralytic symptoms are usually early in syphilis. The affection of the speech and tongue is not present. Epileptic seizures are more common and more liable to be cortical or Jacksonian in character. The expansive delirium is rare. While symptoms of general paresis are not common in connection with the development of gummata or definite gummatous meningitis, there are, on the other hand, instances of paresis which follow syphilitic infection so closely that an etiological connection between the two must be acknowledged. Post mortem in such cases there may be nothing more than a general arterio-sclerosis and diffuse meningo-encephalitis, which may present nothing distinctive, but the lesions, nevertheless, may be caused by

the syphilitic virus. There are certain forms of lead encephalopathy which resemble general paresis, and, considering the association of plumbism with arterio-sclerosis, it is not unlikely that the anatomical substratum of the disease may result from this poison.

Prognosis.—The disease rarely ends in recovery. As a rule the progress is slowly downward and the case terminates in a few years, although it is occasionally prolonged ten or fifteen years.

Treatment.—The only hope of permanent relief is in the cases following syphilis, which should be placed upon large doses of iodide of potassium. Careful nursing and the orderly life of an asylum are the only measures necessary in a great majority of the cases. For sleeplessness and the epileptic seizures bromides may be used. Prolonged remissions, which are not uncommon, are often erroneously attributed to the action of remedies.

VII. TUMORS OF THE BRAIN.

The following are the most common varieties of new growths within the cranium:

(*a*) *Tubercle*, which may form small or large growths, usually multiple. They are most frequent early in life. Three fourths of the cases occur under twenty, and one half of the patients are under ten years of age (Gowers). Of 299 cases of tumor in persons under nineteen collected from various sources by Starr, 152 were tubercle. They are most numerous in the cerebellum and about the base.

(*b*) *Syphiloma* is most commonly found in the hemispheres or about the pons. The tumors are superficial, attached to the arteries or the meninges, and rarely grow to a large size. They may be multiple.

(*c*) *Glioma and Neuroglioma.*—These vary greatly in appearance. They may be firm and hard, almost like an area of sclerosis, or soft and very vascular. They persist remarkably for many years. Klebs has called attention to the occurrence of elements in them not unlike ganglion-cells. Tumors of this character contain "the spinnen" or spider cells; enormous spindle-shaped cells with single large nuclei; cells like the ganglion-cells of nerve-centres with nuclei and one or more processes; and translucent, band-like fibres, tapering at each end, which result from a vitreous or hyaline transformation of the large spindle-cells.

(*d*) *Sarcoma* occurs most commonly in the membranes of the brain and in the pons. It forms some of the largest and most diffusely infiltrating of intracranial growths.

(*e*) *Carcinoma* not infrequently is secondary to cancer in other parts. It is seldom primary. Occasionally cancerous tumors have been found in symmetrical parts of the brain.

(*f*) Other varieties occur, such as fibroid growths, which usually develop from the membranes; bony tumors, which grow sometimes from

Brain cysts. Browning. Jr. N. & Ment Dis. Feb 1883
Conner Am Jr. July 1884

the falx, and psammoma and cholesteatoma. Fatty tumors are occasionally found on the corpus callosum.

(*g*) *Cysts* occur between the membranes and the brain, the result of hæmorrhage or of softening. Porencephalus is a sequence of congenital atrophy or of hæmorrhage, or may be due to a developmental defect. Hydatid cysts will be referred to in the section on parasites.

Symptoms.—(1) **General.**—The following are the most important: *Headache*, either dull, aching, and continuous, or sharp, stabbing, and paroxysmal. It may be diffused over the entire head or limited to the back or front. In the former case it may extend down the neck, and in the latter be accompanied with neuralgic pains in the face. Occasionally the pain may be very localized and associated with tenderness on pressure.

Optic Neuritis.—This occurs in four fifths of all the cases (Gowers). It is usually double, but occasionally is found in only one eye. A growth may develop slowly and attain considerable size without producing optic neuritis. On the other hand, it may occur with a very small tumor, more commonly in a growth at the base.

Vomiting.—This is a common feature, and with headache and optic neuritis makes up the characteristic symptom group of cerebral tumor. An important point is the absence of definite relation to the meals. It may be very obstinate, particularly in growths of the cerebellum and the pons.

Giddiness.—This is often an early symptom. The patient complains of vertigo on rising suddenly or on turning quickly. *Mental Disturbance.* The patient may act in an odd, unnatural manner, or there may be stupor and heaviness. The patient may become emotional or silly, or symptoms resembling hysteria may develop. *Convulsions*, either general and resembling true epilepsy or localized (Jacksonian) in character.

(2) **Localizing Symptoms.**—(*a*) *Central Motor Area.*—The symptoms are either irritative or destructive in character. Irritation in the lower third may produce spasm in the muscles of the face, in the angle of the mouth, or in the tongue. The spasm with tingling may be strictly limited to one muscle group before extending to others, and this Seguin terms the *signal symptom.* The middle third of the motor area contains the centres controlling the arm, and here, too, the spasm may begin in the fingers, in the thumb, in the muscles of the wrist, or in the shoulder. In the upper third of the motor areas the irritation may produce spasm beginning in the toes, in the ankles, or in the muscles of the leg. In many instances the patient can determine accurately the point of origin of the spasm, and there are important sensory disturbances, such as numbness and tingling, which may be felt first at the region affected.

In all cases it is important to determine, first, the point of origin, the *signal symptom;* second, the order or march of the spasm; and third, the subsequent condition of the parts first affected, whether it is a state of paresis or anæsthesia.

Destructive lesions in the motor zone cause paralysis, which is often preceded by local convulsive seizures; there may be a monoplegia, as of the leg, and convulsive seizures in the arm, often due to irritation in these centres. Tumors in the neighborhood of the motor area may cause localized spasms and subsequently, as the centres are invaded by the growth, paralysis occurs. On the left side, growths in the third frontal or Broca's convolution may cause motor aphasia.

(*b*) *Prefrontal Region.*—Neither motor nor sensory disturbance may be present. The general symptoms are often well marked. The most striking feature of growths in this region is mental torpor and gradual imbecility. In its extension downward the tumor may involve on the left side the lower frontal convolution and produce aphasia, or in its progress backward cause irritative or destructive lesions of the motor area.

(*c*) Tumors in the *parieto-occipital lobe* may grow to large size without causing any symptoms. There may be word-blindness and mind-blindness when the angular gyrus is involved, and paraphasia.

(*d*) Tumors of the *occipital lobe* produce hemianopia, and a bilateral lesion may produce blindness. Tumors in this region on the left hemisphere may be associated with word-blindness and mind-blindness.

(*e*) Tumors in the *temporal lobe* may attain a large size without producing symptoms. In their growth they involve the lower motor centres. On the left side involvement of the first and second gyri may be associated with word-deafness.

(*f*) Tumors growing in the neighborhood of the *basal ganglia* produce hemiplegia from involvement of the internal capsule. Limited growths in either nucleus of the corpus striatum do not necessarily cause paralysis. Tumors in the thalamus opticus may also, when small, cause no symptoms, but increasing they may involve the fibres of the optic radiation, producing hemianopia and sometimes hemianæsthesia. Growths in this situation are apt to cause early optic neuritis and, growing into the third ventricle, may cause a distention of the lateral ventricles. In fact, pressure symptoms from this cause and paralysis due to involvement of the internal capsule are the chief symptoms of tumor in and about these ganglia.

Growths in the *corpora quadrigemina* are rarely limited, but most commonly involve the crura cerebri as well. Ocular symptoms are marked. The pupil reflex is lost and there is nystagmus. In the gradual growth the third nerve is involved as it passes through the crus, in which case there will be motor oculi paralysis on one side and hemiplegia on the other, a combination almost characteristic of unilateral crus disease.

(*g*) Tumors of the *pons* and *medulla.* The symptoms are chiefly those of pressure upon the nerves emerging in this region. In disease of the pons the nerves may be involved alone or with the tract. Of 52 cases analyzed by Mary Putnam Jacobi, there were 13 in which the cranial nerves were involved alone, 13 in which the limbs were affected, and 26 in which there was hemiplegia and involvement of the nerves. Twenty-two

In tuberculosis (or syphilis) a growth at the upper and upper aspect of the crus may cause paralysis of the 3rd nerve on one side & ataxic paresis, hypoglossal & limbs on the opposite (Syndrome of Weber)

The features of cerebellar tumor have already been given (p.)

~~Graves. R. of Me Jr. June 1st 1895~~

Paresis of the trunk muscles, as unfit us unable to perform the movements of bending erection & lateral flexion of the trunk may be present. (Nieneyer. J.H. Jackson)
B.m.J. 2.24.94

of the latter had what is known as alternate paralysis—i. e., involvement of the nerves on one side and the limbs on the opposite side. In four cases there were no motor symptoms. A tumor growing in the lower part of the pons usually involves the sixth nerve, producing internal strabismus; the seventh nerve, producing facial paralysis; and the auditory nerve, causing deafness. Conjugate deviation of the eyes to the side opposite that on which there is facial paralysis also occurs.

Tumors of the *medulla* may involve the cranial nerves alone or cause in some instances a combination of hemiplegia with paralysis of the nerves. Signs of irritation in the ninth, tenth, and eleventh nerves are usually present, and produce difficulty in swallowing, irregular action of the heart, irregular respiration, vomiting, and sometimes retraction of the head and neck. The gait may be unsteady or, if there is pressure on the cerebellum, ataxic. Occasionally there are sensory symptoms, numbness, and tingling. Toward the end convulsions may occur.

(*h*) Tumors of the *cerebellum* constitute by far the most important affection of this part. There may be no symptoms whatever if the tumor is confined to one hemisphere and does not involve the middle lobe. When this portion is affected the symptoms are very characteristic, consisting of:

Vertigo, which is more constant in this than in affections of any other region of the brain. This may be due, some believe, to the central relations of the semicircular canals with the cerebellum. The giddiness may be of the most distressing nature.

Headache.—In the analysis by Mary Putnam Jacobi of symptoms in tumors in various parts of the brain headache was relatively much more frequent in tumors of the cerebellum than in any other region.

Cerebellar Ataxia.—The gait is irregular and staggering. In attempting to walk the patient reels to and fro like a drunken man. There may be a tendency to fall to one side, backward, or, less commonly, forward.

Other less constant but suggestive symptoms are the optic neuritis; nystagmus; neuralgic pains in the region of the neck and occiput; pressure symptoms on the medulla, causing vomiting; distention of the lateral ventricles, causing in children hydrocephalus; and, lastly, bilateral rigidity from pressure on the motor paths (Sharkey).

Diagnosis.—From the general symptoms alone the existence of tumor may be determined, for the combination of headache, optic neuritis, and vomiting is distinctive. The localization must be gathered from the consideration of the symptoms above detailed. Mistakes are most likely to occur in connection with uræmia, hysteria, and general paralysis; but careful consideration of all the circumstances of the case usually enables the practitioner to avoid error.

Prognosis.—Syphilitic tumors alone are amenable to treatment. Tuberculous growths occasionally cease to grow and become calcified. The gliomata and fibromata, particularly when the latter grow from the membranes, may last for years. I have described a case of small, hard

glioma, in which the Jacksonian epilepsy persisted for fourteen years. Hughlings Jackson has reported cases of glioma in which the symptoms lasted for over ten years. The more rapidly growing sarcomata usually prove fatal in from six to eighteen months. Death may be sudden, particularly in growths near the medulla; more commonly it is due to coma in consequence of gradual increase in the intracranial pressure.

Treatment.—(*a*) *Medical.*—If there is a suspicion of syphilis the iodide of potassium and mercury should be given. Nowhere do we see more brilliant therapeutical effects than in certain cases of cerebral gummata. The iodide should be given in increasing doses. In tuberculous tumors the outlook is less favorable, though instances of cure are reported, and there is post-mortem evidence to show that the solitary tuberculous tumors may undergo changes and become obsolete. A general tonic treatment is indicated in these cases. The headache usually demands prompt treatment. The iodide of potassium in full doses sometimes gives marked relief. An ice-cap for the head or, in the occipital headache, the application of the Paquelin cautery may be tried. The bromides are not of much use in the headache from this cause, and, as the last resort, morphia must be given. For the convulsions bromide of potassium is of little service.

(*b*) *Surgical.*—Tumors of the brain have been successfully removed by Macewen, Horsley, Keen, and others. The number of cases for operation, however, is small. Four fifths at least of all the cases are probably unsuccessful, or of such a nature as to render an operation fatal. The most advantageous cases are the localized fibromata growing from the dura and only compressing the brain substance, as in Keen's remarkable case. The safety with which the exploratory operation can be made warrants it in all doubtful cases.

VIII. CHRONIC HYDROCEPHALUS.

Definition.—A condition, congenital or acquired, in which there is a great accumulation of fluid within the ventricles of the brain.

The term hydrocephalus has also been applied to the collection of fluid between the cortex of the brain and the skull, known in this situation as *h. externus* or *h. ex vacuo*, a condition common in cases of atrophy of the brain substance, and perhaps caused also by meningeal cysts. A true dropsy, however, of the arachnoid sac probably does not occur.

The cases may be divided into two groups, congenital or infantile, and secondary or acquired.

(1) **Congenital Hydrocephalus.**—The enlarged head may obstruct labor; more frequently the condition is noticed some time after birth. The cause is unknown. It has occurred in several members of the same family.

The anatomical condition in these cases offers no clew to the nature of

the trouble. The lateral ventricles are enormously distended, but the ependyma is usually clear, sometimes a little thickened and granular, and the veins large. The choroid plexuses are vascular, sometimes sclerotic, but often natural-looking. The third ventricle is enlarged, the aqueduct of Sylvius dilated, and the fourth ventricle may be distended. The quantity of fluid may reach several litres. It is limpid and contains a trace of albumen and salts. The changes in consequence of this enormous ventricular distention are remarkable. The cerebral cortex is greatly stretched, and over the middle region the thickness may amount to no more than a few millimetres without a trace of the sulci or convolutions. The basal ganglia are flattened. The skull enlarges, and the circumference of the head of a child of three or four years may reach twenty-five or even thirty inches. The sutures widen, Wormian bones develop in them, and the bones of the cranium become exceedingly thin. The veins are marked beneath the skin. A fluctuation wave may sometimes be obtained, and Fisher's brain murmur may be heard. The orbital plates of the frontal bone are depressed, causing exophthalmos, so that the eyeballs cannot be covered by the eyelids.

Convulsions may occur. The reflexes are increased, the child learns to walk late, and ultimately in severe cases the legs become feeble and sometimes spastic. The mental condition is variable; the child may be bright, but, as a rule, there is some grade of imbecility. The congenital cases usually die within the first four or five years. The process may be arrested and the patient may reach adult life. Cases of this sort are not very uncommon. Even when extreme, the mental faculties may be retained, as in Bright's celebrated patient, Cardinal, who lived to the age of twenty-nine, and whose head was translucent when the sun was shining behind him. Care must be taken not to mistake the rachitic head for hydrocephalus.

(2) **Acquired Chronic Hydrocephalus.**—This is stated to be occasionally primary (idiopathic)—that is to say, it comes on spontaneously in the adult without observable lesion. Dean Swift is said to have died of hydrocephalus, but this seems very unlikely. It is based upon the statement that "he (Mr. Whiteway) opened the skull and found much water in the brain," a condition no doubt of *h. ex vacuo*, due to the wasting associated with his prolonged illness and paralysis. In nearly all cases there is either a tumor at the base of the brain or in the third ventricle, which compresses the venæ Galeni. The passage from the third to the fourth ventricle may be closed, either by a tumor or by parasites. More rarely the foramen of Magendie, through which the ventricles communicate with the cerebrospinal meninges, becomes closed by meningitis. These conditions, occurring in adults, may produce the most extreme hydrocephalus without any enlargement of the head. Even when the tumor begins early in life there may be no expansion of the skull. In the case of a girl aged sixteen, blind from her third year, the head was not unusually large, the ventricles were

enormously distended, and in the Rolandic region the brain substance was only five millimetres in thickness. A tumor occupied the third ventricle. In a case of cholesteatoma of the floor of the third ventricle, in which the symptoms persisted at intervals for eight or nine years, the ventricles were enormously distended without enlargement of the skull. In other instances the sutures separate and the head gradually enlarges.

The symptoms of hydrocephalus in the adult are curiously variable. In the case first mentioned there were early headaches and gradual blindness; then a prolonged period in which she was able to attend to her studies. Headaches again supervened, the gait became irregular and somewhat ataxic. Death occurred suddenly. In the other case there were prolonged attacks of coma with a slow pulse, and on one occasion the patient remained unconscious for more than three months. Gradually progressing optic neuritis without focalizing symptoms, headache, and attacks of somnolence or coma are suggestive symptoms. Cases are rare as a result of meningitis. The only instances I have seen were two which corresponded to the posterior meningitis of Gee and Barlow, in which, with the distention, there was extensive chronic purulent ependymitis.

Treatment.—Very little can be done to relieve hydrocephalus. Medicines are powerless to cause the absorption of the fluid. More rational is the system of gradual compression, with or without the withdrawal of small quantities of the fluid. The compression may be made by means of broad plasters, so applied as to cross each other on the vertex, and another may be placed round the circumference.

Of late years puncture of the ventricles, an operation which had been abandoned, has been revived, particularly by Keen, and in a few cases is justifiable. When pressure symptoms are marked it may be employed with great relief to the headache and removal of the spastic state of the legs. Quincke recommends, and has practised in these cases, as well as in acute hydrocephalus, puncture of the subarachnoid sac between the third and the fourth lumbar vertebræ. At this point the spinal cord cannot be touched. The advantage is a slower removal of fluid and less danger of collapse.

IV. GENERAL AND FUNCTIONAL DISEASES.

I. ACUTE DELIRIUM (*Bell's Mania*).

Definition.—Acute delirium running a rapidly fatal course, with slight fever, and in which post mortem no lesions are found sufficient to account for the disease.

Cases are reported by many old writers under the term brain fever or phrenitis. Bell, at the time Superintendent of the McLean Asylum, de-

but
procedure may be used: He recommends

scribed it* accurately under the designation, "a form of disease resembling some advanced stages of mania and fever."

The disease may set in abruptly or be preceded by a period of irritability, restlessness, and insomnia. The mental symptoms develop with rapidity and may quickly reach a grade of the most intense frenzy. There are the wildest hallucinations and outbreaks of great violence. The patient talks incessantly, but incoherently and unintelligibly. No sleep is obtained, and at last, worn out with the intensity of the muscular movements, the patient becomes utterly prostrated and assumes the sitting or recumbent posture. There may sometimes be definite salaam movements, and in a case which I saw at Westphal's clinic the patient incessantly made motions as if working a pump handle. After a period of intense bodily excitement, lasting for from twenty-four to thirty-six hours or longer, the patient can be examined, and presents the conditions which Bell described as typho-mania. The temperature ranges from 102° to 104°, or even higher. The tongue is dry, the pulse rapid and feeble, and sometimes there are seen on the skin bullæ and pustules, and frequently sores from abrasion and self-inflicted injuries. Toward the close or, according to Spitzka, even during the development of the disease there may be lucid intervals. There may be petechiæ on the skin, and often there is marked congestion of the face and extremities. The duration of the disease is variable. Very acute cases may terminate within a week; others persist for two or even three weeks. The course of the disease is almost uniformly fatal. The anatomical condition is practically negative, or at any rate presents nothing distinctive. There is great venous engorgement of the vessels of the meninges and of the gray cortex. In two cases in which I made a careful microscopic examination of the gray matter there were perivascular exudation and leucocytes in the lymph sheaths and perigangliar spaces. In the inspection of fatal cases of acute delirium careful examination should be made of the lungs and ileum. It should be borne in mind that in a majority of the cases dying in this manner, there is engorgement of the bases of the lungs or even deglutition pneumonia.

The nature of the disease is quite unknown. Some of the cases suggest acute infection. Spitzka thinks that it is due to an autochthonous nerve poison.

Diagnosis.—There are several diseases which may present identical symptoms. As Bell remarks in his paper, the first glance in many cases suggests typhoid fever, particularly when the patient is seen after the violence of the mania subsides. He gives two instances of this which were admitted from a general hospital. Enlargement of the spleen, the occurrence of spots, and the history give clews for the separation of the cases; but there are instances in which it is at first impossible to decide. More-

* American Journal of Insanity, 1849.

over, typhoid fever may set in with the most intense delirium. The existence of fever is the most deceptive symptom, and its combination with delirium and dry tongue so commonly means typhoid fever that it is very difficult to avoid error.

Acute pneumonia may come on with violent maniacal delirium and the pulmonary symptoms may be entirely masked.

Occasionally acute uræmia sets in suddenly with intense mania, and finally subsides into a fatal coma. The condition of the urine and the absence of fever would be important diagnostic features.

The character of the delirium is quite different from that of *mania à potu*. It may be extremely difficult to differentiate acute delirium from certain cases of cortical meningitis, which, however, is usually a secondary affection, occurring in connection with pneumonia or ulcerative endocarditis, or is due to extension from disease of the ear. This sets in more frequently with a chill, and there may be convulsions.

Treatment.—Even though bodily prostration is apt to come on early and be profound, I would not hesitate to advise, in the case of a robust man, free venesection. It is not at all improbable that some of the many cases of mania in which Benjamin Rush let blood with such benefit belonged to this class of affections. Considering its remarkable calming influence in febrile delirium, the cold bath or the cold pack should be employed. Morphia and chloroform may be administered, and hyoscine and the bromides may be tried. Krafft-Ebing states that Solivetti has obtained good results by the use of ergotin. Unfortunately, as asylum reports show, the disease is almost uniformly fatal.

II. PARALYSIS AGITANS

(Parkinson's Disease; Shaking Palsy).

Definition.—A chronic affection of the nervous system, characterized by muscular weakness, tremors, and rigidity.

Etiology.—Men are more frequently affected than women. It rarely occurs under forty, but instances have been reported in which the disease began about the twentieth year. It is by no means an uncommon affection. Direct heredity is rare, but the patients often belong to families in which there are other nervous affections. Among exciting causes may be mentioned exposure to cold and wet, and business worries and anxieties. In some instances the disease has followed directly upon severe mental shock or trauma. Cases have been described after the specific fevers. Malaria is believed by some to be an important factor, but of this there is no satisfactory evidence.

Morbid Anatomy.—No constant lesions have been found. The similarity between certain of the features of Parkinson's disease and those of old age suggest that the affection may depend upon a premature senil-

ity of certain regions of the brain. Our organs do not age uniformly, but in some, owing to hereditary disposition, the process may be more rapid than in others. "Parkinson's disease has no characteristic lesions, but on the other hand it is not a neurosis. It has for an anatomical basis the lesions of cerebro-spinal senility, and which only differ from those of true senility in their early onset and greater intensity." (Dubief.) The important changes are doubtless in the cerebral cortex.

Symptoms.—The disease begins gradually, usually in one or other hand, and the tremor may be either constant or intermittent. With this may be associated weakness or stiffness. At first these symptoms may be present only after exertion. Although the onset is slow and gradual in nearly all cases, there are instances in which it sets in abruptly after fright or trauma. When well established the disease is very characteristic, and the diagnosis can be made at a glance. The four prominent symptoms are tremor, weakness, rigidity, and the attitude.

Tremor.—This may be in the four extremities or confined to hands or feet; the head is not so commonly affected. The tremor is usually marked in the hands, and the thumb and forefinger display the motion made in the act of rolling a pill. At the wrist there are movements of pronation and supination, and less marked of flexion and extension. The upper-arm muscles are rarely involved. In the legs the movement is most evident at the ankle-joint, and less in the toes than in the fingers. Shaking of the head is less frequent, but does occur, and is usually vertical, not rotatory. The rate of oscillation is about five per second. Any emotion exaggerates the movement. The attempt at a voluntary movement may check the tremor (the patient may be able to thread a needle), but it returns with increased intensity. The tremors cease, as a rule, during sleep, but persist when the muscles are at repose. The writing of the patient is tremulous and zigzag.

Weakness.—Loss of power is present in all cases, and may occur even before the tremor, but is not very striking, as tested by the dynamometer, until the late stages. The weakness is greatest where the tremor is most developed. The movements, too, are remarkably slow. There is rarely complete loss of power.

Rigidity may early be expressed in a slowness and stiffness in the voluntary movements, which are performed with some effort and difficulty, and all the actions of the patient are deliberate. This rigidity is in all the muscles, and leads ultimately to the characteristic

Attitude and Gait.—The head is bent forward, the back is bowed, and the arms are held away from the body and are somewhat flexed at the elbow joints. The face is expressionless, and the movements of the lips are slow. The eyebrows are elevated, and the whole expression is beautifully mask-like, the so-called Parkinson's mask. The voice, as pointed out by Bernard, is apt to be shrill and piping, and there is often a hesitancy in beginning a sentence; then the words are uttered with rapidity, as if the

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patient was in a hurry. This is sometimes in striking contrast to the scanning speech of insular sclerosis. The fingers are flexed and in the position assumed when the hand is at rest; in the late stages they cannot be extended. Occasionally there is overextension of the terminal phalanges. The hand is usually turned toward the ulnar side, and the attitude somewhat resembles that of advanced cases of rheumatoid arthritis. In the late stages there are contractures at the elbows, knees, and ankles. The movements of the patient are characterized by great deliberation. He rises from the chair slowly in the stooping attitude, with the head projecting forward. In attempting to walk the steps are short and hurried, and, as Trousseau remarks, he appears to be running after his centre of gravity. This is termed festination or propulsion, in contradistinction to a peculiar gait observed when the patient is pulled backward, when he makes a number of steps and would fall over if not prevented—retropulsion.

The reflexes are normal in most cases, but in a few they are exaggerated.

Of sensory disturbances Charcot has noted abnormal alterations in the temperature sense. The patient may complain of subjective sensations of heat, either general or local—a phenomenon which may be present on one side only and associated with an actual increase of the surface temperature, as much as 6° F. (Gowers). In other instances, patients complain of cold. Localized sweating may be present. The mental condition rarely shows any change.

Variations in the Symptoms.—The tremor may be absent, but the rigidity, weakness, and attitude are sufficient to make the diagnosis. The disease may be hemiplegic in character, involving only one side or even one limb. Usually these are but stages of the disease.

Diagnosis.—In well-developed cases the disease is recognized at a glance. The attitude, gait, stiffness, and mask-like expression are points of as much importance as the oscillations, and usually serve to separate the cases from senile and other forms of tremor. Disseminated sclerosis develops earlier, and is characterized by the nystagmus, and the scanning speech, and does not present the *attitude* so constant in paralysis agitans. The hemiplegic form might be confounded with post-hemiplegic tremor, but the history, the mode of onset, and the greatly increased reflexes would be sufficient to distinguish the two. The Parkinsonian face is of great importance in the diagnosis of the obscure and anomalous forms.

The disease is incurable. Periods of improvement may occur, but the tendency is for the affection to proceed progressively downward. It is a slow, degenerative process and the cases last for years.

Treatment.—There is no method which can be recommended as satisfactory in any respect. Arsenic, opium, and hyoscyamia may be tried, but the friends of the patient should be told frankly that the disease is incurable, and that nothing can be done except to attend to the physical comforts of the patient.

Other Forms of Tremor.

(a) Simple Tremor.—This is occasionally found in persons in whom it is impossible to assign any cause. It may be transient or persist for an indefinite time. It is often extremely slight, and is aggravated by all causes which lower the vitality.

(b) Hereditary Tremor.—C. L. Dana has reported remarkable cases of hereditary tremor. It occurred in all the members of one family, and beginning in infancy it continued without producing any serious changes.

(c) Senile Tremor.—With advancing age tremulousness during muscular movements is extremely common, but is rarely seen under seventy. It is always a fine tremor, which begins in the hands and often extends to the muscles of the neck, causing slight movement of the head.

(d) Toxic tremor is seen chiefly as an effect of tobacco, alcohol, lead, or mercury; more rarely in arsenical or opium poisoning. In elderly men who smoke much it may be entirely due to the tobacco. One of the commonest forms of this is the alcoholic tremor, which occurs only on movement and has considerable range. Lead tremor will be considered in speaking of lead poisoning, of which it constitutes a very important symptom.

(e) Hysterical tremor, which usually occurs under circumstances which make the diagnosis easy, will be considered in the section on hysteria.

III. ACUTE CHOREA

(Sydenham's Chorea; St. Vitus's Dance).

Definition.—A disease chiefly affecting children, characterized by irregular, involuntary contraction of the muscles, a variable amount of psychical disturbance, and a remarkable liability to acute endocarditis.

We shall speak here only of Sydenham's chorea. Senile chorea, chronic chorea, the prehemiplegic and post-hemiplegic forms, and rhythmic chorea are totally different affections.

Etiology.—*Sex.*—Of 554 cases which I have analyzed from the Philadelphia Infirmary for Diseases of the Nervous System, seventy-one per cent were in females and twenty-nine per cent in males. After puberty the percentage in females increases.

Age.—The age incidence in 529 cases was as follows: In the first decade, 301; in the second decade, 248; in the third decade, 10; in the fourth decade, 1; above the fourth decade, 2. In the cases under twenty years the following is the age incidence in the hemidecades: In the first hemidecade, 53; in the second hemidecade, 198; in the third hemidecade, 212; in the fourth hemidecade, 52.

Station.—While the disease affects children of all grades of society, it is more common among the lower classes.

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Station.—While the disease affects children of all grades of society, it is more common among the lower classes.

Race.—As shown by inquiries instituted by Weir Mitchell some years ago, chorea is rare in the negro. No negro child of full birth has been under treatment at the Philadelphia Infirmary. From inquiries made among the medical men who practise in the Indian Territories and in the Indian schools in this country, I find that the disease is unknown in the native races.

Seasonal Relations.—Morris J. Lewis has analyzed 437 separate attacks with reference to this point. Throughout December, January, and February the cases increase There is a fall in April, a rise through May and July, and then a steady fall until October. The cases are most numerous when the mean relative humidity and barometric pressure are low.

Rheumatism.—A causal relationship between rheumatism and chorea has been claimed by many since the time of Bright. The English and French writers maintain the closeness of this connection, and Roger goes so far as to regard the disease in all cases as a manifestation of rheumatism. On the other hand, German authors, as a rule, regard the connection as by no means very close. Discrepancy such as exists between the figures of Steiner, who found only 4 cases of acute rheumatism in 252 cases of chorea, and English writers, such as Dickenson, Barlow, and others, who place the percentage at from fifty to seventy of the cases, can only be explained on the supposition that the connection varies greatly in different localities. Of 554 cases which I have analyzed, in 15·5 per cent there was a history of rheumatism in the family. In 88 cases, 15·8 per cent, there was a history of articular swelling, acute or subacute. In 33 cases there were pains, sometimes described as rheumatic, in various parts, but not associated with joint trouble. If we regard all such cases as rheumatic and add them to those with manifest articular trouble, the percentage is raised to nearly twenty-one.

We find two groups of cases in which acute arthritis is present in chorea. In one, the arthritis antedates by some months or years the onset of the chorea, and does not recur before or during the attack. In the other group, the chorea sets in with or follows immediately upon the acute arthritis. In some instances it is impossible to decide whether the joint trouble or the movements come first. It is difficult to differentiate the cases of irregular pains without definite joint affection. It is probable that many of them are rheumatic, and yet I think it would be a mistake to regard as such all cases in children in which there are complaints of vague pains in the bones or muscles—so-called growing pains. It should never be forgotten, however, that a slight articular swelling may be the sole manifestation of rheumatism in a child—so slight indeed, that the disease may be entirely overlooked. The statistics of the Collective Investigation Committee of the British Medical Association, based upon 439 cases, give twenty-six per cent of antecedent joint affection, and if the cases of vague pains believed to be rheumatic are added, the percentage is raised to thirty-two. In this country rheumatism is not

so common in children as in England. Of the last 144 cases of the Infirmary series, almost every one of which I saw personally, and in which the most minute inquiries were made about rheumatism, there were only 25 cases with articular pains or swelling, and in only 6 had there been acute inflammatory rheumatism. The question may reasonably be asked, Do these articular affections of chorea belong to true rheumatism? Are they not analogous to the joint troubles of scarlet fever, puerperal fever, and gonorrhœa, which no one now regards as truly rheumatic? They have been spoken of by French writers as choreic arthropathies.

Heart-disease.—Endocarditis is believed by some writers to be the cause of the disease. The particles of fibrin and vegetations from the valves pass as emboli to the cerebral vessels. On this view, which we shall discuss later, chorea is the result of an embolic process occurring in the course of a rheumatic endocarditis.

Infectious Diseases.—Scarlet fever with arthritic manifestations may be a direct antecedent. It may be mentioned that a history of this disease occurred in 141 cases, or about twenty-five per cent. Sturges states that a history of previous whooping-cough occurs more frequently in choreic than in other children, but I find no evidence of this in the Infirmary records. With the exception of rheumatic fever, there is no intimate relationship between chorea and the acute diseases incident to childhood. It may be noted in contrast to this that the so-called canine chorea is a common sequel of distemper. Chorea has been known to develop in the course of an acute pyæmia, and to follow gonorrhœa and puerperal fever.

Kinnicutt and others have reported cases of chorea in malarial fevers, but the association was probably accidental, not causal. Anæmia is less often an antecedent than a sequence of chorea, and though cases develop in children who are anæmic and in poor health, this is by no means the rule. Chorea may develop in chlorotic girls at puberty.

Pregnancy.—Chorea may occur during pregnancy—most often during the first five months. It is more common in a first pregnancy, and is rare in women over twenty-five years of age. The disease is usually severe and maniacal symptoms may develop. Occasionally it comes on after an abortion or after delivery at term.

A tendency to the disease is found in certain families. In eighty cases there was a history of attacks of chorea in other members. In one instance both mother and grandmother had been affected. High-strung, excitable, nervous children are specially liable to the disease. *Fright* is considered a frequent cause, but in a large majority of the cases no close connection exists between the fright and the onset of the disease. Occasionally the attack sets in at once. Mental worry, trouble, a sudden grief, or a scolding may apparently be the exciting cause. The strain of *education*, particularly in girls during the third hemidecade, is a most important factor in the etiology of the disease. Bright, intelligent, active-minded girls from ten to fourteen, ambitious to do well at school, often stimulated

in their efforts by teachers and parents, form a large contingent of the cases of chorea in hospital and private practice. Sturges has called special attention to this *school-made* chorea as one serious evil in our modern method of forced education. *Imitation*, which is mentioned as an exciting cause, is extremely rare, and does not appear to have influenced the onset in a single case in the Infirmary records.

The disease may rapidly follow an injury or a slight surgical operation. Reflex irritation was believed to play an important *rôle* in the disease, particularly the presence of worms or genital irritation; but I have met with no instance in which the disease could be attributed to either of these causes. Local spasm, particularly of the face—the habit chorea of Mitchell—may be associated with irritation in the nostrils and adenoid growths in the vault of the pharynx, as pointed out by Jacobi.

It has been claimed by Stevens that *ocular defects* lie at the basis of many cases of chorea, and that with the correction of these the irregular movements disappear. To test the truth of these statements a careful study was made at the Infirmary by De Schweinitz of the condition of the eyes in 50 cases of chorea in children, with the following results: Hypermetropia was present in 23, or forty-six per cent; hypermetropia in one eye and hypermetropic astigmatism in the other in 7, or fourteen per cent; hypermetropic astigmatism in 12, or twenty-four per cent; myopia in 1, or two per cent; myopic astigmatism in 3, or six per cent; mixed astigmatism in 4, or eight per cent. De Schweinitz then adds the cases reported by Stevens and C. S. Bull, of New York, making a total of 227 cases, of which 112 were ametropic and 115 emmetropic. His conclusions are as follows: "Hypermetropia and hypermetropic astigmatism are vastly the preponderating condition in the eyes of choreic children, being found in about seventy-seven per cent of the cases, exactly as hypermetropic refraction is the preponderating condition in childhood, being found in seventy-six per cent of the eyes of children in the elementary schools," and the "evidence, however, seems quite as lacking that hypermetropic refraction is the basal cause of chorea, as it is that the chorea is the cause of the hypermetropia."

The committee of the New York Neurological Society which investigated with great care and impartiality Stevens's claims came to the conclusion that the facts did not warrant their adoption.

Morbid Anatomy and Pathology.—No constant lesions have been found in the nervous system in acute chorea. Vascular changes, such as hyaline transformation, exudation of leucocytes, minute hæmorrhages, and thrombosis of the smaller arteries, have been described.

Embolism of the smaller cerebral vessels has often been found, as might be expected in a disease with which endocarditis is so frequently associated. Based upon this fact, Kirkes, Tuckwell, Hughlings Jackson, and Bastian have supported what is known as the embolic theory of the disease. Endocarditis is by far the most frequent lesion in Sydenham's

The investigations of de Schweinitz show that the ocular defects do not occur in greater proportion in chorea than in other children. A majority of the cases in which operation has been followed by relief have been instances of [illegible] tic, local or general.

Holbers

(To July '94)

I have collected from recent literature the records of 73 recent autopsies; collected from the literature July 1894 there were 62 with Endocarditis.

Osler
1. Chorea & Choreiform affections. London 1894

chorea. With no disease, not excepting rheumatism, is it so constantly associated. In the records of over 110 autopsies, in nearly 100 this condition was mentioned. In the 5 autopsies of which I have notes, in all the mitral valves were affected. The endocarditis is usually of the simple variety, but the ulcerative form has occasionally been described.

We are still far from a solution of all the problems connected with chorea. Unfortunately, the word has been used to cover a series of totally diverse disorders of movement, so that there are still excellent observers who hold that chorea is only a symptom, and is not to be regarded as an etiological unit. The chorea of childhood, the disease which Sydenham described, presents, however, characteristics so unmistakable that it must be regarded as a definite, substantive affection. We cannot discuss fully, but only indicate briefly, certain of the theories which have been advanced with regard to it. The most generally accepted view is that it is a functional brain disorder affecting the nerve-centres controlling the motor apparatus, an instability of the nerve-cells, brought about, one supposes by hyperæmia, another by anæmia, a third by psychical influences, a fourth by irritation, centric or peripheric. Of the actual nature of this derangement we know nothing, nor, indeed, whether the changes are primary and the result of a faulty action of the cortical cells or whether the impulses are secondarily disturbed in their course down the motor path. The predominance of the disease in females, and its onset at a time when the education of the brain is rapidly developing, are etiological facts which Sturges has urged in favor of the view that chorea is an expression of functional instability of the nerve-centres.

The embolic theory originally advanced by Kirkes and supported by the English writers above mentioned has a solid basis of fact, but it is not comprehensive enough, as all of the cases cannot be brought within its limits. There are instances without endocarditis and without, so far as can be ascertained, plugging of cerebral vessels; and there are also cases with extensive endocarditis in which the histological examination of the brain, so far as embolism is concerned, was negative. In two of my post-mortems there were certainly no emboli in the smaller arteries of the branches of the circle of Willis or of the cortex. In the third there was a spot in one corpus striatum of red softening, probably due to an embolus. In favor of the embolic view is the experimental production in animals of chorea by Rosenthal, and later by Money, by injecting fine particles into the carotids of animals.

Lately, as indeed might be expected, a microbic origin has been sought for, and, however improbable such a theory looks at first sight, the case of tetanus gives a warrant, at least, to speculation and investigation in this direction. Nothing definite has yet been determined. From Naunyn's clinic a case is reported with endocarditis and a reddish-brown infiltration of the pia at the base of the brain which proved to be a microbic growth similar in character to those in the vegetations on the heart

valves. Recently, in a fatal case in my wards cultures of a micrococcus were obtained from the blood of the heart, and throughout the brain there were minute foci of hæmorrhage similar to those which occur in pneumonia and other infectious disease associated with endocarditis. In favor of this view it has been urged, as it is impossible to refer the chorea to endocarditis or the endocarditis in all cases to rheumatism, that both have their origin in a common cause, some infectious agent, which is capable also, in persons predisposed, of exciting articular disease. Cases have been reported in scarlet fever with arthritic manifestations, in puerperal fever, and rheumatism, also after gonorrhœa, and such facts are suggestive at least of the association of the disease with infective processes. Possibly, as has been suggested by some writers, the paralytic conditions associated with chorea may be analogous to those which occur in typhoid and certain of the infectious diseases. On the other hand, there are conditions extremely difficult to harmonize with this view. The prominent psychical element is certainly one of the most serious objections, since there can be no doubt that ordinary chorea may rapidly follow a fright or a sudden emotion. It cannot be supposed, too, that the forms associated with reflex irritation, as from the nose and particularly the cases of so-called habit chorea, can be dependent upon infection. We must place these in a separate category, and yet in a long series cases shade so imperceptibly into each other that it is extremely difficult to separate them properly. The question deserves careful study, and the possibility of a special infectious agent has of late been advocated by several writers.

Symptoms.—Three groups of cases may be recognized—the mild, severe, and maniacal chorea.

Mild Chorea.—In this the affection of the muscles is slight, the speech is not seriously disturbed, and the general health not impaired. Premonitory symptoms are shown in restlessness and inability to sit still, a condition well characterized by the term "fidgets." There are emotional disturbances, such as crying spells, or sometimes night-terrors. There may be pains in the limbs and headache. Digestive disturbances and anæmia may be present. A change in the temperament is frequently noticed, and a docile, quiet child may become cross and irritable. After these symptoms have persisted for a week or more the characteristic involuntary movements begin, and are often first noticed at the table, when the child spills a tumbler of water or upsets a plate. There may be only awkwardness or slight incoördination of voluntary movements, or constant irregular clonic spasms. The jerky, irregular character of the movements differentiates them from almost every other disorder of motion. In the mild cases only one hand, or the hand and face, are affected, and it may not spread to the other side.

In the second grade, the *severe form*, the movements become general and the patient may be unable to get about or to feed or undress herself, owing to the constant, irregular, clonic contractions of the various muscle

groups. The speech is also affected, and for days the child may not be able to talk. Often with the onset of the severer symptoms there is loss of power on one side or in the limb most affected.

The third and most extreme form, the so-called maniacal chorea, or *chorea insaniens*, is truly a terrible disease, and may develop out of the ordinary form. A young girl, aged eighteen, was admitted to the Montreal Hospital October 17. She was a waitress at a hotel, and being badly frightened by two men who were fighting, she dropped a tray of dishes which she was carrying. A severe reprimand increased her worry and trouble. The next day she packed her trunk and went home, a distance of thirty miles. Her father insisted that she should return. At this time her hands and arms began to twitch in a violent manner. Five days after the first fright she was admitted. The arms and legs were in constant motion, jerking in all directions. The face also was affected. She was rational, but could scarcely speak. On the night of the 19th she had no sleep, but raved and talked all the time, and the movements were incessant. On the 20th, 21st, and 22d the condition persisted and grew worse. The temperature ranged from 101° to 103°, the tongue became dry and cracked, and she became much exhausted. On the night of the 22d the temperature rose to 105° and death followed, ten days after the onset of the symptoms. These cases are more common in adult women and may develop during pregnancy.

Chorea begins, as a rule, in the hands and arms, then involves the face, and subsequently the legs. The movements may be confined to one side —hemichorea. The attack begins oftenest on the right side, though occasionally it is general from the outset. One arm and the opposite leg may be involved. In nearly one fourth of the cases speech is affected; when slight this is only an embarrassment or hesitancy, but in other instances it becomes an incoherent jumble. In very severe cases the child will make no attempt to speak. The inability is in articulation rather than in phonation. The lips and tongue are concerned in the defect. Occasionally the inspiratory muscles are involved, even when the speech is not at all affected, and sobbing and sighing may result. Paroxysms of panting and of hard expiration may occur, or odd sounds may be produced. As a rule the movements cease during sleep.

A prominent symptom is muscular weakness, usually no more than a condition of paresis. The loss of power is slight, but the weakness may be shown by an enfeebled grip or by a dragging of the leg or limping. In his original account Sydenham refers to the "unsteady movements of one of the legs, which the patient drags." There may be extreme paresis with but few movements—the paralytic chorea of Todd. Occasionally a local paralysis or weakness remains after the attack. Case 229 of the Infirmary series, a lad of ten, had severe general chorea in September, 1880, with considerable loss of power in the legs. Recovery was slow, and when he returned in September, 1883, in a second attack of chorea, there was talipes of the

left foot, which had resulted from paralysis in 1880. In Case 21 a wrist-drop persisted for two years, the result of a palsy which came on with chorea. These are probably instances of peripheral neuritis.

A question of some interest is whether choreic spasms extend to the muscles of organic life. The great gastro-intestinal muscle is never affected. There are no symptoms which can be referred to anomalous contractions of the stomach or bowels. The sphincters act normally. Incontinence of urine occurs occasionally, but it is not noted more frequently, I think, in chorea than in other nervous affections. Spasm of the bronchial muscles is not found even in severe cases, in which the respiratory muscles are involved. The pupils are usually dilated, but no irregular contractions occur. The rapid action and disturbed rhythm of the heart present nothing peculiar to the disease, and there is no support for the view that irregular contractions occur in the papillary muscles.

Heart Symptoms.—*Neurotic.*—As so many of the subjects of chorea are nervous girls, it is not surprising that a common symptom is rapidly acting heart. Any emotional disturbance causes at once a marked increase in the number of the beats, and the actions may become irregular and tumultuous. Irregularity, however, is not so special a feature in chorea as rapidity. The patients seldom complain of pain about the heart.

Hæmic Murmurs.—With anæmia and debility, not uncommon associates of chorea in the third and fourth week, we find a corresponding cardiac condition. The impulse is diffuse, perhaps wavy in thin children. The carotids throb visibly, and in the recumbent posture there may be pulsation in the cervical veins. On auscultation a systolic murmur is heard at the base, perhaps, too, at the apex, soft and blowing in quality.

Endocarditis.—As in rheumatism, so in chorea, acute valvulitis rarely gives evidence of its presence by symptoms. It must be sought, and clinical experience has shown that it is usually associated with murmurs at one or other of the cardiac orifices.

For the guidance of the practitioner the following statements may be made :

(1) In thin, nervous children a systolic murmur of soft quality is extremely common at the base, particularly at the second left costal cartilage, and is probably of no moment.

(2) A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

(3) A murmur of maximum intensity at apex, with rough quality, and transmitted to axilla or angle of scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

(4) When in doubt it is much safer to trust to the evidence of eye

Of 140 cases ~~I found~~ the heart normal in
51; in 17 there was functional disturbance
and in 72 ~~there~~ presented signs of
organic heart disease.

and hand than to that of the ear. If the apex beat is in the normal position, and the area of dulness not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

(5) The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency. Of 110 choreic patients* examined more than two years after the attack, 54 presented signs of organic heart-disease.

(6) Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatism.

Sensory Disturbances.—Pain in the affected limbs is not common. Occasionally there is soreness on pressure. There are cases, usually of hemichorea, in which pain in the limbs is a marked symptom. Weir Mitchell has spoken of these as *painful choreas*. The pain may be quite apart from any arthritic complications. Tingling and pricking sensations and numbness are found occasionally. Anæsthesia is very uncommon. Tender points along the lines of emergence of the spinal nerves or along the course of the nerves of the limbs are rare. The French writers have compared these to the hysterogenic points in hysteria, and have also described in certain cases ovarian tenderness. Headache may be a very troublesome symptom.

Psychical disturbances are common, though in a majority of the cases slight in degree. Irritability of temper, marked wilfulness, and emotional outbreaks may indicate a complete change in the character of the child. There is deficiency in the powers of concentration, the memory is enfeebled, and the aptitude for study is lost. Rarely there is progressive impairment of the intellect with termination in actual dementia. Acute melancholia has been described (Edes). Hallucinations of sight and hearing may occur. Patients may behave in an odd and strange manner and do all sorts of meaningless acts. By far the most serious manifestation of this character is the maniacal delirium, occasionally associated with the very severe cases—*chorea insaniens*. Usually the motor disturbance in these cases is aggravated, but it has been overlooked and patients have been sent to an asylum.

The psychical element in chorea is apt to be neglected by the practitioner. It is always a good plan to tell the parents that it is not the muscles alone of the child which are affected, but that the general irritability and change of disposition, so often found, really form part of the disease.

The condition of the *reflexes* in chorea is usually normal. Sinkler made observations at the Philadelphia Infirmary in 50 cases with the following results: In 26 the knee-jerk was normal, in 15 it was diminished in degree, and in 9 it could not be obtained. Trophic lesions rarely occur

* American Journal of the Medical Sciences, 1887, ii.

in chorea unless, as some writers have done, we regard the joint troubles as arthropathies occurring in the course of a cerebro-spinal disease.

Fever is not, as a rule, present in chorea unless complications exist. There may be the most intense and violent movements without any rise of temperature. I have seen instances, however, in which without apparently any visceral or articular disturbances there was slight daily fever. H. A. Hare states that in monochorea the temperature on the affected side may be elevated; but this is not an invariable rule. Fever is found with an acute arthritis, when there is marked endocarditis or pericarditis, though the former may certainly occur with little if any rise in temperature, and in the cases of maniacal chorea, in which the fever may range from 102° to 104°.

Cutaneous Affections.—These are not very numerous, and in a majority of the cases are probably due to arsenic. There may be an erythematous papular rash. A very interesting condition is the pigmentation which has been found in patients who have been taking arsenic for some time. Herpes zoster occasionally occurs. It was noted twice in the Infirmary records. Certain skin eruptions, usually regarded as rheumatic in character, are not uncommon. Thus, erythema nodosum has been described and I have seen several cases with a purpuric urticaria. There may, indeed, be the more aggravated condition of rheumatic purpura, known as Schönlein's *peliosis rheumatica*. Subcutaneous fibrous nodules, which have been noted by English observers in many cases of chorea, associated with rheumatism, are extremely rare in this country. I have not seen an instance in a choreic patient nor is there a reference in the Infirmary records to a case. This has not been because they were not looked for, as I have seen many instances since my attention was called to them in 1881 by Barlow at the Great Ormond Street Children's Hospital. They are certainly less common in this country than in England. In the chorea returns of the Collective Investigation Committee there were 12 cases out of 439. Cheadle states that they are not uncommon in chorea.

Duration and Termination.—From eight to ten weeks is the average duration of an attack of moderate severity. Cases may be so mild as to get well in two or three weeks; on the other hand, there may be found at every clinic for diseases of the nervous system choreic patients who have been under treatment for three, four, or even six months. Chronic chorea rarely follows the minor disease which we have been considering. The cases described under this designation in children are usually instances of cerebral sclerosis or Friedreich's ataxia; but occasionally an attack which has come on in the ordinary way persists for months or years, and recovery ultimately takes place. A slight grade of chorea, particularly noticeable under excitement, may persist for months in nervous children.

The tendency of chorea to recur has been noticed by all writers since Sydenham first made the observation. Of 410 cases analyzed for this pur-

pose, 240 had one attack, 110 had two attacks, 35 three attacks, 10 four attacks, 12 five attacks, and 3 six attacks. The recurrence is apt to be vernal. Rheumatism seems to favor this tendency; of 60 cases in which there were three or more attacks, there was a history of articular disease in 11, a much higher percentage than in cases with only one or two attacks. The occurrence of heart-disease has been thought to increase this liability, but I think it is the other way—recurrences tend to induce endocarditis and valvular disease. Gowers mentions a case with nine recurrences without history of rheumatism in which there were signs of mitral constriction.

Recovery is the rule in children. The statistics of out-patients' departments are not favorable for determining the mortality. A reliable estimate is that of the Collective Investigation Committee of the British Medical Association, in which 9 deaths were reported among 439 cases, about two per cent.

The paralysis rarely persists. Mental dulness may be present for a time, but usually passes away; permanent impairment of the mind is an exceptional sequence.

Diagnosis.—There are few diseases which present more characteristic features, and in a majority of instances the nature of the trouble is recognized at a glance; but there are several affections in children which may simulate and be mistaken for it.

(*a*) Multiple and diffuse cerebral sclerosis. Cases such as the following are often mistaken for ordinary chorea, and have been described in literature as *chorea spastica*: Nellie P., aged nine years, when two years old had fits which recurred constantly for twenty-one days and persisted on and off with great severity for nine months; she never developed satisfactorily; she learned to talk, but gradually began to have irregular movements. In the ninth year the condition was as follows: Speech hesitating; is unable to sit, stand, or feed herself; can move every muscle of the body, but in an irregular, incoördinate way, which prevents her from using any group of muscles. In attempting to grasp an object the fingers are thrown out in a stiff, spasmodic manner, and she is unable to close them over the object.

In such cases, which are not very uncommon, there are doubtless chronic changes in the cortex. As a rule, the movements are readily distinguishable from those of true chorea, but the simulation is sometimes very close; the onset in infancy, the impaired intelligence, increased reflexes, and in some instances rigidity and the chronic course of the disease, separate them sharply from true chorea.

(*b*) Friedreich's ataxia. Cases of this well-characterized disease were formerly classed as chorea. The slow, irregular, incoördinate movements, the scoliosis, scanning speech, the early talipes, the nystagmus, and the family character of the disease are points which should render the diagnosis easy.

(*c*) In rare cases the paralytic form of chorea may be mistaken for polio-myelitis or, when both legs are affected, for paraplegia of spinal origin; but this can only be the case when the choreic movements are very slight. I have at present under my care a young girl with chorea and loss of power in both legs, who was sent to the hospital as an instance of paraplegia due to spinal disease, but the choreic movements were distinct though slight, and a few days' observation sufficed to render clear the nature of the case.

(*d*) Hysteria may simulate chorea minor most closely, and unless there are other manifestations it may be impossible to make a diagnosis. Most commonly, however, the movements in the so-called hysterical chorea are rhythmic and differ entirely from those of ordinary chorea.

(*e*) As mentioned above, the mental symptoms in maniacal chorea may mask the true nature of the disease and patients have even been sent to the asylum.

Treatment.—Abnormally bright, active-minded children belonging to families with pronounced neurotic taint should be carefully watched from the ages of eight to fifteen and not allowed to overtax their mental powers. So frequently in children of this class does the attack of chorea date from the worry and stress incident to school examinations that the competition for prizes or places should be emphatically forbidden.

The treatment of the attack consists largely in attention to hygienic measures, with which alone, in time, a majority of the cases recover. Parents should be told to scan gently the faults and waywardness of choreic children. The psychical element, strongly developed in so many cases, is best treated by quiet and seclusion. The child should be confined to bed in the recumbent posture and mental as well as bodily quiet enjoined. In private practice this is often impossible, but with well-to-do patients the disease is always serious enough to demand the assistance of a skilled nurse. Toys and dolls should not be allowed at first, for the child should be kept amused without excitement. The rest allays the hyper-excitability and reduces to a minimum the possibility of damage to the valve segments should endocarditis exist. Time and again have I seen very severe cases which had resisted treatment for weeks outside a hospital become quiet and the movements subside after two or three days of absolute rest in bed.

The child should be kept apart from other children and, if possible, from other members of the family, and should see only those persons directly concerned with the nursing of the case. Though irksome and troublesome to carry out, this is an important part of the treatment. In the latter period of the disease daily rubbings may be resorted to with great benefit.

The medicinal treatment of the disease is unsatisfactory; with the exception of arsenic, no remedy seems to have any influence in controlling the progress of the affection. Without any specific action, it certainly does good in many cases, probably by improving the general

It may be given as Martin originally advised. (1813) he began
" with five drops and increased one drop every day
until I ~~found the~~ I might begin to disagree with
the stomach or bowels

nutrition. It is conveniently given in the form of Fowler's solution, and the good effects are rarely seen until maximum doses are taken. Children stand the drug so well that I usually begin with five minims three times a day, and after three days increase the dose by one minim each day. When the dose of fifteen minims is reached, it may be continued for a week, and then again increased, if necessary, every day or two, until physiological effects are manifest. On the occurrence of these the drug should be stopped for three or four days. The practice of resuming the administration with smaller doses is rarely necessary, as tolerance is usually established and we can begin with the dose which the child was taking when the symptoms of saturation occurred. I have frequently given as much as twenty-five minims three times a day. Usually the signs of saturation are trivial but plain, and I have never seen any ill effects from the large doses, but I have heard recently of a case of arsenical neuritis due to the administration of Fowler's solution in chorea.

Of other medicines, strychnine, the zinc compounds, nitrate of silver, bromide of potassium, belladonna, chloral, and especially cimicifuga, have been recommended, and may be tried in obstinate cases.

For its tonic effect electricity is sometimes useful; but it is not necessary as a routine treatment. The question of gymnastics is an important one. Early in the disease, when the movements are active, it is not advisable; but during convalescence carefully graduated exercises are undoubtedly beneficial. It is not well, however, to send a choreic child to a school gymnasium, as the stimulus of the other children and the excitement of the romping, violent play is very prejudicial.

Other points in treatment may be mentioned. It is important to regulate the bowels and to attend carefully to the digestive functions. For the anæmia so often present preparations of iron are indicated.

In the severe cases with incessant movements, sleeplessness, dry tongue, and delirium, the important indication is to procure rest, for which purpose chloral may be freely given, and, if necessary, morphia. Chloroform inhalations may be necessary to subdue the intensity of the paroxysms, but the high rate of mortality in this class of cases illustrates how often our best endeavors are fruitless. The wet pack is sometimes very soothing and should be tried. As these patients are apt to sink rapidly into a low typhoid state with heart weakness, a supporting treatment is required from the outset.

Cases are found now and then which drag on from month to month without getting either better or worse and resist all modes of treatment. Change of air and scene is sometimes followed by rapid improvement, and in these cases the treatment by rest and seclusion should always be given a full trial.

In all cases care should be taken to examine the nostrils, and glaring ocular defects should be properly corrected either by glasses or, if necessary, by operation.

After the child has recovered from the attack, the parents should be warned that return of the disease is by no means infrequent, and is particularly liable to follow overwork at school or debilitating influences of any kind. These relapses are apt to occur in the spring. Sydenham advised purging in order to prevent the vernal recurrence of the disease.

IV. OTHER AFFECTIONS DESCRIBED AS CHOREA.

(*a*) **Chorea Major; Pandemic Chorea.**—The common name, St. Vitus's dance, applied to chorea has come to us from the middle ages, when under the influence of religious fervor there were epidemics characterized by great excitement, gesticulations, and dancing. For the relief of these symptoms, when excessive, pilgrimages were made, and, in the Rhenish provinces, particularly to the Chapel of St. Vitus in Zebern. Epidemics of this sort have occurred also during this century, and descriptions of them among the early settlers in Kentucky have been given by Robertson and Yandell. It was unfortunate that Sydenham applied the term chorea to an affection in children totally distinct from this chorea major, which and is in reality an hysterical manifestation under the influence of religious excitement.

(*b*) **Habit Spasm (Habit Chorea); Convulsive Tic** (of the French).

Two groups of cases may be recognized under the designation of habit spasm—one in which there ~~is~~ are simply localized spasmodic movements, and the other in which, in addition to this, there are explosive utterances and psychical symptoms, a condition to which French writers have given the name *tic convulsif*.

(1) *Habit Spasm.*—This is found chiefly in childhood, most frequently in girls from seven to fourteen years of age (Mitchell). In its simplest form there is a sudden, quick contraction of certain of the facial muscles, such as rapid winking or drawing of the mouth to one side, or the neck muscles are involved and there are unilateral movements of the head. The head is given a sudden, quick shake, and at the same time the eyes wink. A not infrequent form is the shrugging of one shoulder. The grimace or movement is repeated at irregular intervals, and is much aggravated by emotion. A short inspiratory sniff is not an uncommon symptom. The cases are found most frequently in children who are "out of sorts," or who have been growing rapidly, or who have inherited a tendency to neurotic disorders. Allied to or associated with this are some of the curious tricks of children. A boy at my clinic was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the forefinger. Hartley Coleridge is said to have had a somewhat similar trick, only he bit his arm. In all these cases the habits of the child should be examined carefully, the nose and vault of the pharynx thoroughly inspected, and the eyes accurately

Or A = the fixed idea about words, - onomatomania [12]

[12] See Arnaut & Marandon. Arch. 4/5. 1892. Obsession about words
11-1 certain sounds & words designated, bête & even budget on the
stomach & were indigestible.

tested. As a rule the condition is transient, and after persisting for a few months or longer gradually disappears. Occasionally a local spasm persists —twitching of the eyelids, or the facial grimace.

(2) *Tic Convulsif* (*Gilles de la Tourette's Disease*).—This remarkable affection, often mistaken for chorea, more frequently for habit spasm, is really a psychosis allied to hysteria, though in certain of its aspects it has the features of monomania. The disease begins, as a rule, in young children, occurring as early as the sixth year, though it may develop after puberty. There is usually a markedly neurotic family history. The special features of the complaint are:

(*a*) Involuntary muscular movements, usually affecting the facial or brachial muscles, but in aggravated cases all the muscles of the body may be involved and the movements may be extremely irregular and violent.

(*b*) Explosive utterances, which may resemble a bark or an inarticulate cry. A word heard may be mimicked at once and repeated over and over again, usually with the involuntary movements. To this the term *echolalia* has been applied. A much more distressing disturbance in these cases is *coprolalia*, or the use of bad language. A child of eight or ten may shock its mother and friends by constantly using the word *damn* when making the involuntary movements, or by uttering all sorts of obscene words. Occasionally actions are mimicked—*echokinesis*.

(*c*) Associated with some of these cases are curious mental disturbances; the patient becomes the subject of a form of obsession or a fixed idea. I was consulted recently about a young girl in whom the spasms were very slight, amounting only to twitching of the eyes and slight jerking of the shoulder, but who had a most pronounced grade of the fixed idea known as *arithmomania*. Almost every action, even the most trifling, was preceded by the counting of a certain number of figures. Before she went to bed she had to tap her heel upon the side of the bedstead a certain number of times; before drinking the tumbler had to be rotated eight or ten times, and then when set down again the same act was repeated. Before opening the door a certain number of knocks had to be given. The greatest difficulty was experienced in getting her to brush her hair, as it took her so long to count the necessary number of figures before she began. In other cases the fixed idea takes the form of the impulse to touch objects. According to Guinon, who has written an exhaustive article upon it in the Dictionnaire Encyclopédique, the prognosis is bad.

The disease is well marked and readily distinguished from ordinary chorea. The movements have a larger range and are explosive in character. Tourette regards the coprolalia as the most distinctive feature of the disease.

(*c*) **Saltatoric Spasm** (*Lata; Myriachit; Jumpers*).—Bamberger has described a disease in which when the patient attempted to stand there were strong contractions in the leg muscles, which caused a jumping or

springing motion. This occurs only when the patient attempts to stand. The affection has occurred in both men and women, more frequently in the former, and the subjects have usually shown marked neurotic tendencies. In many cases the condition has been transitory; in others it has persisted for years. Remarkable affections similar to this in certain points occur as a sort of endemic neurosis. One of the most striking of these occurs among the "jumping Frenchmen" of Maine and Canada. As described by Beard and Thornton, the subjects are liable on any sudden emotion to jump violently and utter a loud cry or sound, and will obey any command or imitate any action without regard to its nature. The condition of echolalia is present in a marked degree. The "jumping" prevails in certain families.

A very similar disease prevails in parts of Russia and in Java, where it is known by the names of myriachit and lata, the chief feature of which is mimicry by the patient of everything he sees or hears.

(*d*) **Chronic Chorea** (*Huntingdon's Chorea*).—An affection characterized by irregular movements, disturbance of speech, and gradual dementia. It is frequently hereditary. The disease has no connection with Sydenham's chorea, and it is unfortunate that the term was applied to it. It was described by Huntingdon, of Pomeroy, Ohio, at the time a practitioner on Long Island, and he gave in three brief paragraphs the salient points in connection with the disease—namely, the hereditary nature, the association with psychical troubles, and the late onset—between the thirtieth and fortieth years. The disease seems common in this country, and many cases have been reported by Clarence King, Sinkler, and others.* I have seen it in two Maryland families within the past two years. Under the term chronic chorea may be grouped the hereditary form and the cases which come on without family disposition, either at middle life or, more commonly, in the aged—senile chorea. It is doubtful whether the cases in children with chronic choreiform movements, often with mental weakness and spastic condition of the legs, should go into this category.

The hereditary character of the disease is very striking, and it has been traced through four or five generations. Huntingdon's father and grandfather, also physicians, had treated the disease in the family which he described. An identical affection occurs without any hereditary disposition. The age of onset is late, rarely before the thirtieth or the thirty-fifth year.

The symptoms are very characteristic. The irregular movements are usually first seen in the hands, and the patient has slight difficulty in performing delicate manipulations or in writing. When well established the movements are disorderly, irregular, incoördinate rather than choreic, and have not the sharp, brusque motion of Sydenham's chorea. In the face there are slow, involuntary grimaces. In a well-developed case the gait is irregular, swaying, and somewhat like that of a drunken man. The speech

* For complete literature, see Huet, de la Chorée Chronique, Paris, 1889.

and Oppenheim & Hoppe have described in two cases a miliary disseminated encephalitis of the cortical & subcortical regions, particularly of the motor zone.

Paramyelonus multiplex. Here much the

is slow and difficult, the syllables are badly pronounced and indistinct, but not definitely staccato. The mental impairment is a gradual enfeeblement, leading finally to dementia. At first the patient may be emotional.

Very few post-mortems have been made. No characteristic lesions have been found. Atrophy of the convolutions, chronic meningo-encephalitis, and vascular changes have usually been present, the conditions which one would expect to find in a chronic dementia. These existed in an autopsy which I have on one of my cases. The affection is evidently a neuro-degenerative disorder, and has no connection with the simple chorea of childhood.

(*e*) **Rhythmic or Hysterical Chorea.**—This is readily recognized by the rhythmical character of the movements. It may affect the muscles of the abdomen, producing the salaam convulsion, or involve the sterno-mastoid, producing a rhythmical movement of the head, or the psoas, or any group of muscles. In its orderly rhythm it resembles the canine chorea.

V. INFANTILE CONVULSIONS (*Eclampsia*).

Convulsive seizures similar to those of epilepsy are not infrequent in children and in adults. The fit may indeed be identical with epilepsy, from which the condition differs in that when the cause is removed there is no tendency for the fits to recur. Occasionally, however, the convulsions in children continue and develop into true epilepsy.

Etiology.—A convulsion in a child may be due to many causes, all of which lead to an unstable condition of the nerve-centres, permitting of sudden, excessive and temporary nervous discharges. The following are the most important of them:

(1) Debility, resulting usually from gastro-intestinal disturbance. Convulsions frequently supervene toward the close of an attack of entero-colitis and recur, sometimes proving fatal. Morris J. Lewis has shown that the death rate in children from eclampsia rises steadily with that of gastro-intestinal disorders.

(2) Peripheral irritation. Dentition alone is rarely a cause of convulsions, but is often one of several factors in a feeble, unhealthy infant. The greatest mortality from convulsions is during the first six months, before the teeth really cut through the gums. Other irritative causes are the overloading of the stomach with indigestible food. It has been suggested that some of these cases are toxic, owing to the absorption of poisonous ptomaines. Worms, to which convulsions are so frequently attributed, probably have little influence. Among other sources possible are phimosis and otitis.

(3) Rickets. The observation of Sir William Jenner upon the association of rickets and convulsions has been amply confirmed. The spasms may be laryngeal, the so-called child-crowing, which, though convulsive in

nature, can scarcely be considered with eclampsia. The influence of this condition is more apparent in Europe than in this country, although rickets is a common disease, particularly among the colored people. Spasms, local or general, in rickets are probably associated with the condition of debility and malnutrition and with cranio-tabes.

(4) Fever. In young children the onset of the infectious diseases is frequently with convulsions, which often take the place of a chill in the adult. It is not known upon what they depend. Scarlet fever, measles, and pneumonia are most often preceded by convulsions.

(5) Congestion of the brain. That extreme engorgement of the blood-vessels may produce convulsions is shown by their occasional occurrence in severe whooping-cough, but their rarity in this disease really indicates how small a part mechanical congestion plays in the production of fits.

(6) Severe convulsions usher in or accompany many of the serious diseases of the nervous system in children. In more than fifty per cent of the cases of infantile hemiplegia the affection follows severe convulsions. They less frequently precede a spinal paralysis. They occur with meningitis, tuberculous or simple, and with tumors and other lesions of the brain.

And, lastly, convulsions may occur immediately after birth and persist for weeks or months. In such instances there has probably been meningeal hæmorrhage or serious injury to the cortex.

The most important question is the relation of convulsions in children to true epilepsy. In Gowers's figures of 1,450 cases of epilepsy, the attacks began in 180 during the first three years of life. Of 460 cases of epilepsy in children which I have analyzed, in 187 the fits began within the first three years. Of the total list the greatest number, 74, was in the first year. In nearly all these instances there was no interruption in the convulsions. That convulsions in early infancy are necessarily followed by epilepsy in after life is certainly a mistake.

Symptoms.—The attack may come on suddenly without any warning; more commonly it is preceded by a stage of restlessness, accompanied by twitching and perhaps grinding of the teeth. It is rarely so complete in its stages as true epilepsy. The spasm begins usually in the hands, most commonly in the right hand. The eyes are fixed and staring or are rolled up. The body becomes stiff and breathing is suspended for a moment or two by tonic spasm of the respiratory muscles, in consequence of which the face becomes congested. Clonic convulsions follow, the eyes are rolled about, the hands and arms twitch, or are flexed and extended in rhythmical movements, the face is contorted, and the head is retracted. The attack gradually subsides and the child sleeps or passes into a state of stupor. Following indigestion the attack may be single, but in rickets and intestinal disorders it is apt to be repeated. Sometimes the attacks follow each other with great rapidity, so that the child never rouses but dies

in a deep coma. If the convulsion has been limited chiefly to one side there may be slight paresis after recovery, or in instances in which the convulsions usher in infantile hemiplegia, when the child arouses one side is completely paralyzed. During the fit the temperature is often raised. Death rarely occurs from the convulsion itself, except in debilitated children or when the attacks recur with great frequency. In the so-called hydrocephaloid state in connection with protracted diarrhœa convulsions may close the scene.

Diagnosis.—Coming on when the subject is in full health, the attack is probably due either to overloaded stomach, to some peripheral irritation, or occasionally to trauma. Setting in with high fever and vomiting, it may indicate the onset of an exanthem, or occasionally be the primary symptom of encephalitis, or whatever the condition is which causes infantile hemiplegia. When the attack is associated with debility and with rickets the diagnosis is easily made. The carpopedal spasms and pseudo-paralytic rigidity which are often associated with rickets, laryngismus stridulus, and the hydrocephaloid state are usually confined to the hands and arms and are intermittent and usually tonic. The convulsions associated with tumor or which follow infantile hemiplegia are usually at first Jacksonian in character. After the second year convulsive seizures which come on irregularly without apparent cause and recur while the child is apparently in good health are likely to prove true epilepsy.

Prognosis.—Convulsions play an important part in infantile mortality. In Morris J. Lewis's table of deaths in children under ten, 8·5 per cent were ascribed to convulsions. West states that 22·35 per cent of deaths under one year are caused by convulsions, but this is too high an estimate for this country. In chronic diarrhœa convulsions are usually of ill omen. Those ushering in fevers are rarely serious, and the same may be said of the fits associated with indigestion and peripheral irritation.

Treatment.—Every source of irritation should be removed. If associated with indigestible food, a prompt emetic should be given, followed by an enema. The teeth should be examined, and if the gum is swollen, hot, and tense, it may be lanced; but never if it looks normal. When seen at first, if the paroxysm is severe, no time should be lost by giving a hot bath, but chloroform should be given at once, and repeated if necessary. A child is so readily put under chloroform and with such a small quantity that this precedure is quite harmless and saves much valuable time. The practice is almost universal of putting the child into a warm bath, and if there is fever the head may be douched with cold water. The temperature of the bath should not be above 95° or 96°. The very hot bath is not suitable, particularly if the fits are due to indigestion. After the attack an ice-cap may be placed upon the head. If there is much irritability, particularly in rickets and in severe diarrhœa, small doses of

opium will be found efficacious. When the convulsions recur after the child comes from under the influence of chloroform it is best to place it rapidly under the influence of opium, which may be given as morphia hypodemically, in doses of from one twenty-fifth to one thirtieth of a grain for a child of one year. Other remedies recommended are chloral by enema, in five-grain doses, and nitrite of amyl. After the attack has passed the bromides are useful, of which five to eight grains may be given in a day to a child a year old. Recurring convulsions, particularly if they come on without special cause, should receive the most thorough and careful treatment with bromides. When associated with rickets the treatment should be directed to improving the general condition.

VI. EPILEPSY.

Definition.—An affection of the nervous system characterized by attacks of unconsciousness, with or without convulsions.

The transient loss of consciousness without convulsive seizures is known as *petit mal;* the loss of consciousness with general convulsive seizures is known as *grand mal.* Localized convulsions, occurring usually without loss of consciousness, are known as epileptiform, or more frequently as Jacksonian or cortical epilepsy.

Etiology.—*Age.*—In a large proportion of all cases the disease begins before puberty. Of the 1,450 cases observed by Gowers, in 422 the disease began before the tenth year, and three fourths of the cases began before the twentieth year. Of 460 cases of epilepsy in children which I have analyzed * the age of onset in 427 was as follows: First year, 74; second year, 62; third year, 51; fourth year, 24; fifth year, 17; sixth year, 18; seventh year, 19; eighth year, 23; ninth year, 17; tenth year, 27; eleventh year, 17; twelfth year, 18; thirteenth year, 15; fourteenth year, 21; fifteenth year, 34. Arranged in hemidecades the figures are as follows: From the first to the fifth year, 229; from the fifth to the tenth year, 104; from the tenth to the fifteenth year, 95. These figures illustrate in a striking manner the early onset of the disease in a large proportion of the cases. It is well always to be suspicious of epilepsy developing in the adult, for in a majority of such cases the convulsions are due to a local lesion.

Sex.—No special influence appears to be discoverable in this relation, certainly not in children. Of 433 cases in my tables, 232 were males and 203 were females, showing a slight predominance of the male sex. After puberty unquestionably, if a large number of cases are taken, the males

* Three hundred and nine cases from the records from the Philadelphia Infirmary for Diseases of the Nervous System, 126 cases at the Elwyn Institution for Feeble-minded Children, and 25 from the records of my neurological clinic at the Johns Hopkins Hospital.

are in excess. The figures of Sieveking and Reynolds show that the disease is rather more prevalent in females than in males.

Heredity.—Much stress has been laid upon this by many authors as an important predisposing cause, and the statistics collected give from nine to over forty per cent. Gowers gives thirty-five per cent for his cases, which have special value apart from other statistics embracing large numbers of epileptics in that they were collected by him in his own practice. In our figures it appears to play a minor *rôle.* In the Infirmary list there were only 31 cases in which there was a history of marked neurotic taint, and only three in which the mother herself had been epileptic. In the Elwyn cases, as might be expected, the percentage is larger. Of the 126 there was in 32 a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity. It is interesting to note that in this group, in which the question of heredity is carefully looked into, there were only two in which the mother had had epilepsy, and not one in which the father had been affected. Indeed, I was not a little surprised to find in the list of my cases that hereditary influences played so small a part. I have heard this opinion expressed by certain French physicians, notably Marie, who in writing also upon the question takes strong grounds against heredity as an important factor in epilepsy.

While, then, it may be said that direct inheritance is comparatively uncommon, the children of neurotic families in which neuralgia, insanity, and hysteria prevail are more liable to fall victim to the disease.

Chronic alcoholism in the parents is regarded by many as a potent predisposing factor in the production of epilepsy. Echeverria has analyzed 572 cases bearing upon this point and divided them into three classes, of which 257 cases could be traced directly to alcohol as a cause; 126 cases in which there were associated conditions, such as syphilis and traumatism; 189 cases in which the alcoholism was probably the result of the epilepsy. Figures equally strong are given by Martin,* who found in 150 insane epileptics 83 with a marked history of parental intemperance. Of the 126 Elwyn cases, in which the family history on this point was carefully investigated, in the majority of instances alcoholism seems not to have existed to any marked degree in the parents, a definite statement being found in only four of the cases.

Syphilis.—This in the parents is probably less a predisposing than an actual cause of epilepsy, which is the direct outcome of local cerebral manifestations. There is no reason for recognizing a special form of syphilitic epilepsy. On the other hand, convulsive seizures due to acquired syphilitic disease of the brain are very common.

Of exciting causes fright is believed to be important, but is less so, I think, than is usually stated. Trauma is present in a certain number of instances. An important group depends upon a local disease of the brain

* Annales Médicales Psychologiques, 1879.

existing from childhood, as seen in the post-hemiplegic epilepsy. Occasionally cases follow the infectious fevers. Masturbation has been stated to be a special cause, but its influence is probably overrated. A large group of convulsive seizures allied to epilepsy are due to some toxic agent, as in lead-poisoning and in uræmia. Great stress was laid upon reflex causes, such as dentition and worms, the irritation of a cicatrix, some local affection, such as adherent prepuce, or a foreign body in the ear or the nose. In many of these cases the fits cease after the removal of the cause, so that there can be no question of the association between the two. In others the attacks persist. Genuine cases of reflex epilepsy are, I believe, rare. A remarkable instance of it occurred at the Philadelphia Infirmary for Diseases of the Nervous System in the case of a man with a testis in the inguinal canal, pressure upon which would cause a typical fit. Removal of the organ was followed by cure.

Epilepsy has been thought to be associated with disturbance of the heart's action, and some have spoken of a special cardiac epilepsy, particularly in cases in which there is palpitation or slowing of the action prior to the onset. Epileptic seizures may occur during the passage of a gall-stone or occasionally during the removal of pleuritic fluid. Indigestion and gastric troubles are extremely common in epilepsy, and in many instances the eating of indigestible articles seems to precipitate an attack.

An attempt to associate genuine epilepsy with eye-strain has signally failed.

Symptoms.—(1) **Grand Mal.**—Preceding the fits there is usually a localized sensation, known as an *aura*, in some part of the body. This may be somatic, in which the feeling comes from some particular region in the periphery, as from the finger or hand, or is a sensation felt in the stomach or about the heart. The peripheral sensations preceding the fit are of great value, particularly those in which the aura always occurs in a definite region, as in one finger or toe. It is the equivalent of the signal symptom in a fit from a brain tumor. The varieties of these sensations are numerous. The epigastric sensations are most common. In these the patient complains of an uneasy sensation in the epigastrium or distress in the intestines, or the sensation may not be unlike that of heart-burn and may be associated with palpitation. These groups are sometimes known as pneumogastric auræ or warnings.

Of psychical auræ one of the most common, as described by Hughlings Jackson, is the vague, dreamy state, a sensation of strangeness or sometimes of terror. The auræ may be associated with special senses, of which the visual are the most common, consisting of flashes of light or sensations of color; less commonly, distinct objects are seen. The auditory auræ consist of noises in the ear, odd sounds, musical tunes, or occasionally voices. Olfactory and gustatory auræ, unpleasant tastes and odors, are rare.

Occasionally the fit may be preceded not by an aura, but by certain

Alcohol.

(Mr Fishers case recovery
Mr Platt.

movements; the patient may turn round rapidly or run with great speed for a few minutes, the so-called epilepsia procursiva. In one of the Elwyn cases the lad stood on his toes and twirled with extraordinary rapidity, so that his features were scarcely recognizable. At the onset of the attack the patient may give a loud scream or yell, the so-called epileptic cry. The patient drops as if shot, making no effort to guard the fall. In consequence of this, epileptics frequently injure themselves, cutting the face or head or burning themselves. In the attack, as described by Hippocrates, "the patient loses his speech and chokes, and foam issues from the mouth, the teeth are fixed, the hands are contracted, the eyes distorted, he becomes insensible, and in some cases the bowels are affected. And these symptoms occur sometimes on the left side, sometimes on the right, and sometimes on both." The fit may be described in three stages:

(*a*) *Tonic Spasm.*—The head is drawn back or to the right, and the jaws are fixed. The hands are clinched and the legs extended. This tonic contraction affects the muscles of the chest, so that respiration is impeded and the initial pallor of the face changes to a dusky or livid hue. The muscles of the two sides are unequally affected, so that the head and neck are rotated or the spine is twisted. The feet are extended and the knees and hip-joint are flexed. The arms are usually flexed at the elbows, the hand at the wrist, and the fingers are tightly clinched in the palm. This stage lasts only a few seconds, and then the

(*b*) *Clonic stage* begins. The muscular contractions become intermittent; at first tremulous or vibratory, they gradually become more rapid and the limbs are jerked and tossed about violently. The muscles of the face are in constant clonic spasm, the eyes roll, the eyelids are opened and closed convulsively. The movements of the muscles of the jaw are very forcible and strong, and it is at this time that the tongue is apt to be caught between the teeth and lacerated. The cyanosis, marked at the end of the tonic stage, gradually lessens. A frothy saliva, which may be blood-stained, escapes from the mouth. The fæces and urine may be discharged involuntarily. The duration of this stage is variable. It rarely lasts more than one or two minutes. The contractions become less violent and the patient gradually sinks into the condition of

(*c*) *Coma.* The breathing is noisy or even stertorous, the face congested, but no longer intensely cyanotic. The limbs are relaxed and the unconsciousness is profound. After a variable time the patient can be aroused, but if left alone he sleeps for some hours and then awakes, complaining only of slight headache or mental confusion.

In some cases one attack follows the other with great rapidity and consciousness is not regained. This is termed the *status epilepticus*, an exceptional condition, in which the patient may die of exhaustion consequent

upon the repeated attacks. In it the temperature is usually elevated. After the attack the reflexes are sometimes absent; more frequently they are increased and the ankle clonus can usually be obtained.

The state of the urine is variable, particularly as regards the solids. The quantity is usually increased after the attack, and albumen is not infrequently present.

Post-epileptic symptoms are of great importance. The patient may be in a trance-like condition, in which he performs actions of which subsequently he has no recollection. More serious are the attacks of mania, in which the patient is often dangerous and sometimes homicidal. It is held by good authorities that an outbreak of mania may be substituted for the fit. And, lastly, the mental condition of an epileptic patient is often seriously impaired, and profound defects are common.

Paralysis, which rarely follows the epileptic fit, is usually hemiplegic and transient.

Slight disturbances of speech also may occur; in some instances forms of sensory aphasia.

The attacks may occur at night, and a person may be epileptic for years without knowing it. As Trousseau truly remarks, when a person tells us that in the night he has incontinence of urine and awakes in the morning with headache and mental confusion, and complains of difficulty in speech owing to the fact that he has bitten his tongue; if, also, there are on the skin of the face and neck purpuric spots, the probability is very strong indeed that he is subject to nocturnal epilepsy.

(2) **Petit Mal.**—This is epilepsy without the convulsions. The attack consists of transient unconsciousness, which may come on at any time, accompanied or unaccompanied by a feeling of faintness and vertigo. Suddenly, for example, at the dinner table, the subject stops talking and eating, the eyes become fixed, and the face slightly pale. Anything which may have been in the hand is usually dropped. In a moment or two consciousness is regained and the patient resumes conversation as if nothing had happened. In other instances there is slight incoherency or the patient performs some almost automatic action. He may begin to undress himself and on returning to consciousness find that he has partially disrobed. In other attacks the patient may fall without convulsive seizures. A definite aura is rare. Though transient, unconsciousness and giddiness are the most constant manifestations of *petit mal;* there are many other equivalent manifestations, such as sudden jerkings in the limbs, sudden tremor, or a sudden visual sensation. Gowers mentions no less than seventeen different manifestations of *petit mal.*

After the attack the patient may be dazed for a few seconds and perform certain automatic actions, which may seem to be volitional. As mentioned, undressing is a common action, but all sorts of odd actions may be performed, some of which are awkward or even serious. One of my patients after an attack was in the habit of tearing anything he could lay

hands on, particularly books. Violent actions have been committed and assaults made, frequently giving rise to questions which come before the courts. This condition has been termed masked epilepsy, or *epilepsia larvata*.

In a majority of the cases of *petit mal* convulsions finally occur, at first slight, but ultimately the *grand mal* becomes well developed, and the attacks may then alternate.

(3) **Jacksonian Epilepsy.**—This is also known as cortical, symptomatic, or partial epilepsy. It is distinguished from the ordinary epilepsy by the important fact that consciousness is retained. The attacks are usually the result of irritative lesions in the motor zone, though there are probably also sensory equivalents of this motor form. In a typical attack the spasm begins in a limited muscle group of the face, arm, or leg. The zygomatic muscles, for instance, or the thumb may twitch, or the toes may first be moved. Prior to the twitching the patient may feel a sensation of numbness or tingling in the part affected. The spasm extends and may involve the muscles of one limb only or of the face. The patient is conscious throughout and watches, often with interest, the march of the spasm.

The onset may be slow, and there may be time, as in a case which I have reported, for the patient to place a pillow on the floor, so as to be as comfortable as possible during the attack. The spasms may be localized for years, but there is a great risk that the partial epilepsy may become general. The condition is due, as a rule, to an irritative lesion in the motor zone. Thus of 107 cases analyzed by Roland, there were 48 of tumor, 21 instances of inflammatory softening, 14 instances of acute and chronic meningitis, and 8 cases of trauma. The remaining instances were due to hæmorrhage or abscess, or were associated with sclerosis cerebri. Two other conditions may be mentioned, which may cause typical Jacksonian epilepsy—namely, uræmia and progressive paralysis of the insane. A considerable number of the cases of Jacksonian epilepsy are found in children following hemiplegia, the so-called post-hemiplegic epilepsy. The convulsions usually begin on the affected side, either in the arm or leg, and the fit may be unilateral and without loss of consciousness. Ultimately they become more severe and general.

Diagnosis.—In major epilepsy the suddenness of the attack, the abrupt loss of consciousness, the order of the tonic and clonic spasm, and the relaxation of the sphincters at the height of the attack are distinctive features. The convulsive seizures due to uræmia are epileptic in character and usually readily recognized by the existence of greatly increased tension and the condition of the urine. Practically in young adults hysteria causes the greatest difficulty, and may closely simulate true epilepsy. The following table from Gowers's work draws clearly the chief differences between them:

	Epileptic.	Hysteroid.
Apparent cause.....	none.	emotion.
Warning.....	any, but especially unilateral or epigastric auræ.	palpitation, malaise, choking, bilateral foot aura.
Onset.....	always sudden.	often gradual.
Scream.....	at onset.	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone.	rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting.....	tongue.	lips, hands, or other people and things.
Micturition.....	frequent.	never.
Defecation.....	occasional.	never.
Talking	never.	frequent.
Duration	a few minutes.	more than ten minutes, often much longer.
Restraint necessary..	to prevent accident.	to control violence.
Termination.....	spontaneous.	spontaneous or induced (water, etc.).

Recurring epileptic seizures in a person over thirty who has not had previous attacks is always suggestive of organic disease. According to H. C. Wood, whose opinion is supported by that of Fournier, in nine cases out of ten the condition is due to syphilis.

Petit mal must be distinguished from attacks of syncope, and the vertigo of Menière's disease, of a cardiac lesion, and of indigestion. In these cases there is no actual loss of consciousness, which forms a characteristic though not an invariable feature of *petit mal.*

Jacksonian epilepsy has features so distinctive and peculiar that it is at once recognized. It is by no means easy, however, always to determine upon what the spasm depends. Irritation in the motor centres may be due to a great variety of causes, among which tumors and localized meningo-encephalitis are the most frequent; but it must not be forgotten that in uræmia localized epilepsy may occur. The most typical Jacksonian spasms also are not infrequent in general paresis of the insane.

Prognosis.—This may be given to-day in the words of Hippocrates: "The prognosis in epilepsy is unfavorable when the disease is congenital, and when it endures to manhood, and when it occurs in a grown person without any previous cause. . . . The cure may be attempted in young persons, but not in old."

Death during the fit rarely occurs, but it may happen if the patient falls into the water or if the fit comes on while he is eating. Occasionally the fits seem to stop spontaneously. This is particularly the case in the epilepsy in children which has followed the convulsions of teething or of the fevers. Frequency of the attacks and marked mental disturbance are unfavorable indications. Hereditary predisposition is apparently of no moment in the prognosis. The outlook is better in males than in females. The post-hemiplegic epilepsy is rarely arrested. Of the cases coming on

in adults, those due to syphilis and to local affections of the brain allow a more favorable prognosis.

Treatment.—*General.*—In the case of children the parents should be made to understand from the outset that epilepsy in the great majority of cases is an incurable affection, so that the disease may interfere as little as possible with the education of the child. The subjects need firm but kind treatment. Indulgence and yielding to caprices and whims are followed by weakening of the moral control, which is so necessary in these cases. The disease does not incapacitate a person for all occupation. It is much better for epileptics to have some definite pursuit. There are many instances in which they have been persons of extraordinary mental and bodily vigor; as, for example, Julius Cæsar and Napoleon. One of the most distressing features in epilepsy is the gradual mental impairment which follows in a certain number of cases. If such patients become extremely irritable or show signs of violence they should be placed under supervision in an asylum. Marriage should be forbidden to epileptics. During the attack a cork or bit of rubber should be placed between the teeth and the clothes should be loosened. The patient should be in the recumbent posture. As the attack usually passes off with rapidity, no special treatment is necessary, but in cases in which the convulsion is prolonged a few whiffs of chloroform or nitrite of amyl or a hypodermic of a quarter of a grain of morphia may be given.

Dietetic.—The old authors laid great stress upon regimen in epilepsy. The important point is to give the patient a light diet at fixed hours, and on no account to permit overloading of the stomach. Meat should not be given more than once a day. There are cases in which animal food seems injurious. A strictly vegetable diet has been warmly recommended. The patient should not go to sleep until the completion of gastric digestion.

Medicinal.—The bromides are the only remedies which have a special influence upon the disease. Either the sodium or potassium salt may be given. Sodium bromide is probably less irritating and is better borne for a long period. It may be given in milk, in which it is scarcely tasted. In all instances the dilution should be considerable. In adults it is well taken in soda water or in some mineral water. The dose for an adult should be from half a drachm to a drachm and a half daily. As Seguin recommends, it is often best to give but a single dose daily, about four to six hours before the attacks are most likely to occur. For instance, in the case of nocturnal epilepsy a drachm should be given an hour or two after the evening meal. If the attack occurs early in the morning, the patient should take a full dose when he awakes. When given three times a day it is best given after meals. Each case should be carefully studied to determine how much bromide should be used. The individual susceptibility varies and some patients require more than others. Fortunately, children take the drug well and stand proportionately larger doses than

adults. Saturation is indicated by certain unpleasant effects, particularly drowsiness, mental torpor, and gastric and cardiac distress. Loss of palate reflex is one of the earliest indications that the system is under the influence of the bromides, and is a condition which should be attained. A very unpleasant feature is the development of acne, which, however, is no indication of bromism. Seguin states that the tendency to this is much diminished by giving the drug largely diluted in alkaline waters and administering from time to time full doses of arsenic. To be effectual the treatment should be continued for a prolonged period and the cases should be incessantly watched in order to prevent bromism. The medicine should be continued for at least two years after the cessation of the fits; indeed, Seguin recommends that the reduction of the bromides should not be begun until the patient has been three years without any manifestations. Written directions should be given to the mother or to the friends of the patient, and he should not himself be held responsible for the administration of the medicine. A book should be provided in which the daily number of attacks and the amount of medicine taken should be noted.

Among other remedies which have been recommended as controlling epilepsy are chloral, cannabis indica, zinc, nitroglycerin, and borax. Nitroglycerin is sometimes advantageous in *petit mal*, but is not of much service in the major form. To be beneficial it must be given in full doses, from two to five minims of the one per cent solution, and increased until the physiological effects are produced. Counter-irritation is rarely advisable. When the aura is very definite and constant in its onset, as from the hand or from the toe, a blister about the part or a ligature tightly applied may stop the oncoming fit. In children, care should be taken that there is no source of peripheral irritation. In boys, adherent prepuce may occasionally be the cause. The irritation of teething, the presence of worms, and foreign bodies in the ears or nose have been associated with epileptic seizures.

The subjects of a chronic and, in most cases, a hopelessly incurable disease, epileptic patients form no small portion of the unfortunate victims of charlatans and quacks, who prescribe to-day, as in the time of the father of medicine, "purifications and spells and other illiberal practices of like kind."

Surgical.—In Jacksonian epilepsy the propriety of surgical interference is universally granted. It is questionable, however, whether in the epilepsy following hemiplegia, considering the anatomical condition, it is likely to be of any benefit. In idiopathic epilepsy, when the fit starts in a certain region—the thumb, for instance—and the signal symptom is invariable, the centre controlling this part may be removed. This procedure has been practised by Macewen, Horsley, Keen, and others, but time alone can determine its value. The traumatic epilepsy, in which the fit follows fracture, is much more hopeful.

The addition of belladonna to the bromide is warmly recommended by Black of Glasgow. Pot. brom. half an ounce. Tinctura belladonnæ 3 gr. infusi gentianæ co. ad. 8 ounces—tablespoonful three times a day.

New remedy. Will try.

The operation, *per se*, appears in some cases to have a curative effect. Thus of 50 cases of trephining for epilepsy in which nothing abnormal was found to account for the symptoms, 25 were reported as cured and 18 as improved.* The operations have not been always on the skull, and White has collected an interesting series in which various surgical procedures have been resorted to, often with curative effect, such as ligation of the carotid artery, castration, tracheotomy, excision of the superior cervical ganglia, incision of the scalp, circumcision, etc.

VII. MIGRAINE (*Hemicrania; Sick Headache*).

Definition.—A paroxysmal affection characterized by severe headache, usually unilateral, and often associated with disorders of vision.

Etiology.—The disease is frequently hereditary and has occurred through several generations. Women and the members of neurotic families are most frequently attacked. It is an affection from which many distinguished men have suffered and have left on record an account of the disease, notably the astronomer Airy. Edward Liveing's work is the standard authority upon which most of the subsequent articles have been based. A gouty or rheumatic taint is present in many instances. Sinkler has called special attention to the frequency of reflex causes. Migraine has long been known to be associated with uterine and menstrual disorders. Many of the headaches from eye-strain are of the hemicranial type. Brunton refers to caries of the teeth as a cause of these headaches, even when not associated with toothache. Cases have been described in connection with adenoid growths in the pharynx, and particularly with abnormal conditions of the nose. Many of the attacks of severe headaches in children are of this nature, and the eyes and nostrils should be examined with great care. Sinkler refers to a case in a child of two years, and Gowers states that a third of all the cases begin between the fifth and tenth years of age. The direct influences inducing the attack are very varied. Powerful emotions of all sorts are the most potent. Mental or bodily fatigue, digestive disturbances, or the eating of some particular article of food may be followed by the headache. The paroxysmal character is one of the most striking features, and the attacks may recur on the same day every week, every fortnight, or every month.

Symptoms.—Premonitory signs are present in many cases, and the patient can tell when an attack is coming on. Remarkable prodromata have been described, particularly in connection with vision. Apparitions may appear—visions of animals, such as mice, dogs, etc. Transient hemianopia or scotoma may be present. In other instances there is spasmodic action of the pupil on the affected side, which dilates and contracts

* J. William White, Curative Effects of Operations *per se*, Annals of Surgery, 1891.

alternately, the condition known as *hippus*. Frequently the disturbance of vision is only a blurring, or there are balls of light, or zigzag lines, or the so-called fortification spectra (teichopsia), which may be illuminated with gorgeous colors. Disturbances of the other senses are rare. Numbness of the tongue and face and occasionally of the hand may occur with tingling. More rarely there are cramps or spasms in the muscles of the affected side. Transient aphasia has also been noted. Some patients show marked psychical disturbance, either excitement or, more commonly, mental confusion or great depression. Dizziness occurs in some cases. The headache follows a short time after the prodromal symptoms have appeared. It is cumulative and expansile in character, beginning as a localized small spot, which is generally constant either on the temple or forehead or in the eyeball. It is usually described as of a penetrating, sharp, boring character. At first unilateral, it gradually spreads and involves the side of the head, sometimes the neck, and the pains may pass into the arm. In other cases both sides are affected. Nausea and vomiting are common symptoms. If the attack comes on when the stomach is full, vomiting usually gives relief. Vaso-motor symptoms may be present. The face, for instance, may be pale, and there may be a marked difference between the two sides. Subsequently the face and ear on the affected side may become a burning red from the vaso-dilator influences. The pulse may be slow. The temporal artery on the affected side may be firm and hard, and in a condition of arterio-sclerosis—a fact which has been confirmed anatomically by Thoma. Few affections are more prostrating than migraine, and during the paroxysm the patient may scarcely be able to raise the head from the pillow. The slightest noise or light aggravates the condition.

The duration of the entire attack is variable. The severer forms usually incapacitate the person for at least three days. In other instances the entire attack is over in a day. The disease recurs for years, and in cases with a marked hereditary tendency may persist throughout life. In women the attacks often cease after the climacteric, and in men after the age of fifty. Two of the greatest sufferers I have known, who had recurring attacks every few weeks from early boyhood, now have complete freedom.

The nature of the disease is unknown. Liveing's view, that it is a nerve storm or form of periodic discharge from certain sensory centres and is related to epilepsy, has found much favor. According to this view, it is the sensory equivalent of a true epileptic attack. Mollendorf, Latham, and others regard it as a vaso-motor neurosis, and hold that the early symptoms are due to vaso-constrictor and the later symptoms to vaso-dilator influences. The fact of the development of arterio-sclerosis in the arteries of the affected side is a point of interest bearing upon this view.

Treatment.—The patient is fully aware of the causes which precipitate an attack. Avoidance of excitement, regularity in the meals, and moderation in diet are important rules. The treatment should be directed

toward the removal of the conditions upon which the attacks depend. In children much may be done by watchfulness and care on the part of the mother in regulating the bowels and watching the diet of the child. Errors of refraction should be adjusted. On no account should such children be allowed to compete in school for prizes. A prolonged course of bromides sometimes proves successful. If anæmia is present, iron and arsenic should be given. When the arterial tension is increased a course of nitroglycerin may be tried. Not too much, however, should be expected of the preventive treatment of migraine. It must be confessed that in a very large proportion of the cases the headaches recur in spite of all we can do. During the paroxysm the patient should be kept in bed and absolutely quiet. If the patient feels faint and nauseated, a small cup of hot, strong coffee or twenty drops of chloroform give relief. Cannabis indica is probably the most satisfactory remedy. Seguin recommends a prolonged course of the drug. Antipyrin, antifebrin, and phenacetin have been much used of late. When given early, at the very outset of the paroxysm, they are sometimes effective. The doses which have been recommended of antifebrin and antipyrin are often dangerous, and I have seen in a case of migraine unpleasant collapse symptoms follow a twenty-five-grain dose of antipyrin which the patient had taken on her own responsibility. Smaller, repeated doses are more satisfactory. Of other remedies, caffeine, in five-grain doses of the citrate, nux vomica, and ergot have been recommended. Electricity does not appear to be of much service.

VIII. NEURALGIA.

Definition.—A painful affection of the nerves, due either to functional disturbance of their central or peripheral extremities or to neuritis in their course.

Etiology.—Members of neuropathic families are most subject to the disease. It affects women more than men. Children are rarely attacked. Of all causes, debility is the most frequent. It is often the first indication of an enfeebled nervous system. The various forms of anæmia are frequently associated with neuralgia. It may be a prominent feature at the onset of certain acute diseases, particularly typhoid fever. Malaria is believed to be a potent cause, but it has not been shown that neuralgia is more frequent in malarial districts, and the error has probably arisen from regarding periodicity as a special manifestation of paludism. It occasionally occurs in malarial cachexia. Exposure to cold is a cause in very susceptible persons. Reflex irritation, particularly from carious teeth, may induce neuralgia of the fifth nerve. The disease occurs sometimes in rheumatism, gout, lead poisoning, and diabetes.

Symptoms.—Before the onset of the pain there may be uneasy sensations, sometimes tingling in the part which will be affected. The pain

is localized to a certain group or division of nerves, usually affecting one side. The pain is not constant, but paroxysmal, and is described as stabbing, burning, or darting in character. The skin may be exquisitely tender in the affected region, particularly in certain points along the course of the nerve, the so-called tender points. Movements, as a rule, are painful. Trophic and vaso-motor changes may accompany the paroxysm; the skin may be cool, and subsequently hot and burning, occasionally local œdema or erythema occurs. More remarkable still are the changes in the hair, which may become blanched (canities), or even fall out. Fortunately, such alterations are rare. Twitchings of the muscles, or even spasms, may be present during the paroxysm. After lasting a variable time—from a few minutes to many hours—the attack subsides. Recurrence may be at definite intervals—every day at the same hour, or at intervals of two, three, or even seven days. Occasionally the paroxysms develop only at the catamenia. This periodicity is quite as marked in non-malarial as in malarial regions.

Clinical Varieties, depending on the Nerve Groups affected.—(1) *Trifacial Neuralgia; Tic Douloureux; Prosopalgia.*—All the branches are rarely involved together. The ophthalmic is most often affected, but in severe attacks the pains, though more intense in one division, radiate over the other branches. At the outset there may be hyperæsthesia of the skin and sensitiveness of the mucous membrane. Pressure is painful at the points of emergence of the nerve trunk, and where the nerves enter the muscles. Sometimes in addition, as Trousseau pointed out, there are pains at the occipital protuberance and in the upper cervical spines. When the ophthalmic division is affected the eye may weep and the conjunctivæ are injected and painful. In the upper maxillary division there is a tender point where the nerve leaves the infraorbital canal, and the pain is specially marked along the upper teeth. In the lower branches, which are more frequently involved, there are painful points along the auriculo-temporal nerve and the pain radiates in the region of the ear along the lower jaw and teeth. The movements of mastication and speaking may be painful. Salivation is not uncommon. Herpes may occur about the eye or the lips. In protracted cases there may be atrophy or induration of the skin. Some of the forms of facial neuralgia are of frightful intensity and the recurring attacks render the patient's life almost insupportable.

(2) *Cervico-occipital neuralgia* involves the posterior branches of the first four cervical nerves, particularly the inferior occipital, at the emergence of which there is a painful point about half-way between the mastoid process and the first cervical vertebra. It may be caused by cold, and these nerves are often affected in cervical caries.

(3) *Cervico-brachial neuralgia* involves the sensory nerves of the brachial plexus, particularly in the cubital division. When the circumflex nerve is involved the pain is in the deltoid. The pain is most commonly

about the shoulder and down the course of the ulnar nerve. There is usually a marked tender point upon this nerve at the elbow. This form rarely follows cold, but more frequently results from rheumatic affections of the joints, and trauma.

(4) *Neuralgia of the phrenic nerve* is rare. It is sometimes found in pleurisy and in pericarditis. The pain is chiefly at the lower part of the thorax on a line with the insertion of the diaphragm, and here may be painful points on deep pressure. Full inspiration is painful, and there is great sensitiveness on coughing or in the performance of any movement by which the diaphragm is suddenly depressed.

(5) *Intercostal Neuralgia.*—Next to the *tic douloureux* this is the most important form. It is most frequent in women and very common in hysteria and anæmia. The pain in caries and aneurism is felt in the intercostal nerves. They are also the seat of the intense pain in inflammation of the pleura. The pain is often constant and exaggerated by movements. Pleurodynia is supposed by some to be local intercostal neuralgia, confined to one spot, usually along the course or at the exit of the nerves. Herpes zoster or zona occurs with the most aggravated form of intercostal neuralgia. The pain usually precedes the eruption, which consists of a series of pearly vesicles, which take two or three days to develop and gradually disappear. The eruption may occur without much pain. The most distressing feature in the complaint is the persistence in the pain after the eruption has subsided. The eruption and the neuralgia are in reality manifestations of neuritis. Changes have been found in the nerves and in the ganglia of the posterior roots. The pain of zona may persist indefinitely, and it has been known to be so intractable that in despair the person has committed suicide.

(6) *Lumbar Neuralgia.*—The affected nerves are the posterior fibres of the lumbar plexus, particularly the ilio-scrotal branch. The pain is in the region of the iliac crest, along the inguinal canal, in the spermatic cord, and in the scrotum or labium majus. The affection known as irritable testis, probably a neuralgia of this nerve, may be very severe and accompanied by syncopal sensations.

(7) *Coccydynia.*—This is regarded as a neuralgia of the coccygeal plexus. It is most common in women, and is aggravated by the sitting posture. It is very intractable, and may necessitate the removal of the coccyx, an operation, however, which is not always successful. Neuralgias of the nerves of the leg have already been considered.

(8) *Neuralgias of the Nerves of the Feet.*

Painful Heel.—Both in women and men there may be about the heel severe pains which interfere seriously with walking—the pododynia of S. D. Gross. There may be little or no swelling, no discoloration, and no affection of the joints. The pain is usually most severe over the heel; sometimes in a very limited spot, sometimes in the line of the metatarso-phalangeal joint. Probably this painful affection depends upon many

different conditions. It may be associated with rheumatism or gout, and with certain occupations—persons who have to stand for a long time on their feet. In other instances it occurs with flat-foot.

Plantar Neuralgia.—This is often associated with a definite neuritis, such as follows typhoid fever, and has been seen in an aggravated form in caisson disease (Hughes). The pain may be limited to the tips of the toes or to the ball of the great toe. Numbness, tingling, and hyperæsthesia or sweating may occur with it. Following the cold-bath treatment in typhoid fever it is not uncommon for patients to complain of great sensitiveness in the toes.

Erythromelalgia.—Under this term Weir Mitchell described a condition which is associated with great pain in the heel or in the sole of the foot, with vascular changes, either an acute hyperæmia or cyanosis. Some of the cases should unquestionably be regarded as Raynaud's disease.

(9) *Visceral Neuralgias.*—The more important of these have already been referred to in connection with the cardiac and the gastric neuroses. They are most frequent in women, and are constant accompaniments of neurasthenia and hysteria. The pains are most common in the pelvic region, particularly about the ovaries. Nephralgia is of great interest, for, as has already been mentioned, the symptoms may closely simulate those of stone.

Treatment.—Causes of reflex irritation should be carefully removed. The neuralgia, as a rule, recurs unless the general health improves; so that tonic and hygienic measures of all sorts should be employed. Often a change of air or surroundings will relieve a severe neuralgia. I have known obstinate cases to be cured by a prolonged residence in the mountains, with an out-of-door life and plenty of exercise. Of general remedies, iron is often a specific in the cases associated with chlorosis and anæmia. Arsenic, too, is very beneficial in these forms, and should be given in ascending doses. The value of quinine has been much overrated. It probably has no more influence than any other bitter tonic, except in the rare instances in which the neuralgia is definitely associated with malarial poisoning. Strychnine, cod-liver oil, and phosphorus are also advantageous. Of remedies for the pain, the new analgesics should first be tried—antipyrin, antifebrin, and phenacetin—for they are sometimes of service. Morphia should be given with great caution, and only after other remedies have been tried in vain. On no consideration should the patient be allowed to use the hypodermic syringe. Gelsemium is highly recommended. Of nervine stimulants, valerian and ether, which often act well together, may be given. Alcohol is a valuable, though dangerous, remedy, and should not be ordered for women. In the trifacial neuralgia nitroglycerin in large doses may be tried. Aconitia in doses of from one two-hundredth to one one-hundred-and-fiftieth of a grain may be tried. In gouty and rheumatic subjects cannabis indica and cimicifuga are recommended with the lithium salts.

Metatarsalgia, Morton's (Thomas G.) "painful affection of the fourth metatarso-phalangeal articulation" is a peculiar ~~affection~~ and very trying disorder, seen most frequently in women, & usually in one foot. Morton regards it as due to a pinching of the metatarsal nerve. The disease rarely gets well without operation

Of local applications, the thermo-cautery is invaluable, particularly in zona and the more chronic forms of neuralgia. Acupuncture may be used, or aquapuncture, the injection of distilled water beneath the skin. Chloroform liniment, camphor and chloral, menthol, the oleates of morphia, atropia, and belladonna used with lanolin may be tried. Freezing over the tender point with ether spray is sometimes successful. The continuous current may be used. The sponges should be warm, and the positive pole should be placed near the seat of the pain. The strength of the current should be such as to cause a slight tingling or burning, but not pain.

The surgical treatment of intractable neuralgia embraces nerve stretching and excision. The latter is the most satisfactory, but too often the pain returns.

IX. PROFESSIONAL SPASMS; OCCUPATION ~~NEUROSES.~~ ?

The continuous and excessive use of the muscles in performing a certain movement may be followed by an irregular, involuntary spasm or cramp, which may completely check the performance of the action. The condition is found most frequently in writers, hence the term writer's cramp or scrivener's palsy; but it is also common in piano and violin players and in telegraph operators. The spasms occur in many other persons, such as milkmaids, weavers, and cigarette-rollers.

The most common form is writer's cramp, which is much more frequent in men than in women. Of 75 cases of impaired writing power reported by Poore, all of the instances of undoubted writer's cramp were in men. Morris J. Lewis states that in this country, in the telegrapher's cramp, women, who are employed a great deal in telegraphy, are much less frequently affected (only 4 out of 43 cases). Persons of a nervous temperament are more liable to the disease. Occasionally it follows slight injury.

Gowers states that in a majority of the cases a faulty method of writing has been employed, using either the little finger or the wrist as the fixed point. Persons who write from the middle of the forearm or from the elbow are rarely affected.

No anatomical changes have been found. The most reasonable explanation of the disease is that it results from a deranged action of the nerve centres presiding over the muscular movements involved in the act of writing, a condition which has been termed irritable weakness. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this

lessened resistance be carried too far, there is an increase and irregular discharge of nerve energy, which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves." (Gay.)

Symptoms.—These may be described under five heads (Lewis).

(*a*) *Cramp* or *Spasm.*—This is often an early symptom and most commonly affects the forefinger and thumb ; or there may be a combined movement of flexion and adduction of the thumb, so that the pen may be twisted from the grasp and thrown to some distance. Weir Mitchell has described a lock-spasm, in which the fingers become so firmly contracted upon the pen that it cannot be removed.

(*b*) *Paresis* and *Paralysis.*—This may occur with the spasm or alone. The patient feels a sense of weakness and debility in the muscles of the hand and arm and holds the pen feebly. Yet in these circumstances the grasp of the hand may be strong and there may be no paralysis for ordinary acts.

(*c*) *Tremor.*—This is most commonly seen in the forefinger and may be a premonitory symptom of atrophy. It is not an important symptom, and is rarely sufficient to produce disability.

(*d*) *Pain.*—Abnormal sensations, particularly a tired feeling in the muscles, are very constantly present. Actual pain is rare, but there may be irregular shooting pains in the arm. Numbness or soreness may exist. If, as sometimes happens, a subacute neuritis develops, there may be pain over the nerves and numbness or tingling in the fingers.

(*e*) *Vasomotor Disturbances.*—These may occur in severe cases. There may be hyperæsthesia. Occasionally the skin becomes glossy, or there is a condition of local asphyxia resembling chilblains. In attempting to write, the hand and arm may become flushed and hot and the veins increased in size. Early in the disease the electrical reactions are normal, but in advanced cases there may be diminution of faradic and sometimes increase in the galvanic irritability.

Diagnosis.—A well-marked case of writer's cramp or palsy could scarcely be mistaken for any other affection. Care must be taken to exclude the existence of any cerebro-spinal disease, such as progressive muscular atrophy or hemiplegia. The physician is sometimes consulted by nervous persons who fancy they are becoming subject to the disease and complain of stiffness or weakness without displaying any characteristic features.

Prognosis.—The course of the disease is usually chronic. If taken in time and if the hand is allowed perfect rest, the condition may improve rapidly, but too often there is a strong tendency to recurrence. The patient may learn to write with the left hand, but this also may after a time be attacked.

Treatment.—Various prophylactic measures have been advised. As mentioned, it is important that a proper method of writing be adopted. Gowers suggests that if all persons wrote from the shoulder writer's cramp would practically not occur. Various devices have been invented for relieving the fatigue, but none of them are very satisfactory. The use of the type-writer has diminished very much the frequency of scrivener's palsy. Rest is essential. No measures are of value without this. Massage and manipulation, when combined with systematic gymnastics, give the best results. Poore recommends the galvanic current applied to the muscles, which are at the same time rhythmically exercised.

The nutrition of the patients is apt to be much impaired, and cod-liver oil, strychnia, and other tonics will be found advantageous. Local applications are of little benefit. Tenotomy and nerve-stretching have been abandoned.

X. TETANY.

Definition.—An affection characterized by peculiar tonic spasms, either paroxysmal or continued, of the extremities.

Etiology.—The disease occurs under very different conditions. Four forms may be recognized.

(*a*) Epidemic tetany, also known as rheumatic tetany. In certain parts of the continent of Europe the disease has prevailed widely, particularly in the winter season. Von Jaksch, who has described an epidemic form occurring in young men of the working classes, sometimes with [illegible] only two or three cases and again [illegible]

(*b*) A majority of the cases are found in association with debility following lactation and chronic diarrhœa, or in the malnutrition of children. From its frequency in nursing women Trousseau called it nurse's contracture. It may occur during pregnancy. It has been found as a sequence of the acute fevers, and in some epidemics [illegible] have occurred.

(*c*) Tetany may follow removal of the thyroid gland. Thirteen cases, for example, followed eighty-eight operations on enlarged thyroid in Billroth's clinic, and six of these proved fatal. James Stewart has reported an instance in which with the tetany there were symptoms of myxœdema, and no trace of the thyroid gland. Removal of the thyroid in dogs is also followed by tetany.

(*d*) And, lastly, there is a [illegible] associated with dilatation of the stomach, particularly after the stomach has been washed out. A case has been reported in this [illegible]

On the continent tetany is an extremely rare disease. In the discussion on Stewart's case at the Association of American Physicians, Washington, 1891, Wm. H. Welch stated that he had seen but two instances in

B E Vaughan has reported ~~(N. Y. Med Jr. 1843)~~ an epidemic of tetanus in Italians in N. Y. ~~cases~~ occurred in one family.

It is a rare complication; (H. M. Thomas)
Only about 12 cases are on record altogether.

Case of dilated stomach. Gorge Americaine. no hard tetany
the day before his death.

Griffith has collected 72 cases, in which however he has included
cases of carpo-pedal spasm in children

Treatment.—Various prophylactic measures have been advised. As mentioned, it is important that a proper method of writing be adopted. Gowers suggests that if all persons wrote from the shoulder writer's cramp would practically not occur. Various devices have been invented for relieving the fatigue, but none of them are very satisfactory. The use of the type-writer has diminished very much the frequency of scrivener's palsy. Rest is essential. No measures are of value without this. Massage and manipulation, when combined with systematic gymnastics, give the best results. Poore recommends the galvanic current applied to the muscles, which are at the same time rhythmically exercised.

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(*b*) A majority of the cases are found in association with debility following lactation and chronic diarrhœa, or in the malnutrition of rickets. From its occurrence in nursing women Trousseau called it nurse's contracture. It may also occur during pregnancy. It has been found as a sequence of the acute fevers, and in some typhoid epidemics many cases have occurred.

(*c*) Tetany may follow removal of the thyroid gland. Thirteen cases, for example, followed seventy-eight operations on enlarged thyroid in Billroth's clinic, and six of them proved fatal. James Stewart has reported an instance in which with the tetany there were symptoms of myxœdema, and no trace of the thyroid gland. Removal of the thyroid in dogs has also been followed by tetany.

(*d*) And, lastly, there is a form of fatal tetany which is associated with dilatation of the stomach, particularly after the organ has been washed out. A case has been reported in this country by F. T. Miles.

On this continent tetany is an extremely rare disease. In the discussion on Stewart's case at the Association of American Physicians, Washington, 1889, Weir Mitchell stated that he had seen but two instances in

his long and varied experience, while Pepper had seen but one case, and that was in a child.

The nature of the disease is unknown, but it probably depends upon the action of some toxic agent on the motor-nerve cells.

Symptoms.—In cases associated with general debility or in children with rickets the spasm is limited to the hands and feet. The fingers are bent at the metacarpo-phalangeal joint, extended at the terminal joints, pressed close together, and the thumb is contracted in the palm of the hand. The wrist is flexed, the elbows are bent, and the arms are folded over the chest. In the lower limbs the feet are extended and the toes adducted. The muscles of the face and neck are less commonly involved, but in severe cases there may be trismus, and the angles of the mouth are drawn out. The skin of the hands and feet is sometimes tense and œdematous. The spasms are usually paroxysmal and last for a variable time. In children the attack may pass off in a few hours. In some of the severer chronic cases in adults the stiffness and contracture may continue or even increase for many days, and the attack may last as long as two weeks. In the acute cases the temperature may be elevated and the pulse quickened. In the severe paroxysms there may be involvement of the muscles of the back and of the thorax, inducing dyspnœa and cyanosis. Two additional features, valuable in diagnosis, are present. The irritability of the nerves is enormously increased both during the period of tetany and subsequently. Thus a minimal strength of current necessary to produce a contracture during the quiescent period is sufficient during the attack to cause a distinct tetanic contraction. The second point is the so-called Trousseau's phenomenon: pressure on the larger arteries, sometimes on the nerve trunk, will excite the spasm, which continues while the pressure is kept up.

Diagnosis.—The disease is readily recognized. It is a mistake to call instances of carpo-pedal spasm of children true tetany. It is common to find in rickety children or in cases of severe gastro-intestinal catarrh a transient spasm of the fingers or even of the arms. By many authors these are considered cases of mild tetany, and there are all grades in rickety children between the simple carpo-pedal spasm and the condition in which the four extremities are involved; but it is well, I think, to limit the term tetany to the severer affection.

With true tetanus the disease is scarcely ever confounded, as the commencement of the spasm in the extremities, the attitude of the hands, and the etiological factors are very different. Hysterical contractures are usually unilateral.

Except in the cases associated with dilated stomach and those which follow thyroidectomy the prospect of recovery is good.

Treatment.—In the case of children the condition with which the tetany is associated should be treated. Baths and cold sponging are recommended and often relieve the spasm as promptly as in child-crowing.

There may be an association between disturbed thyroid function and tetany.

Trousseau's symptom "So long as the attack is not over, the paroxysms may be reproduced at will .... This is effected by simply compressing the affected parts, either in the direction of their principal nerve-trunks or over their blood vessels, so as to impede the venous or arterial circulation" (Trousseau)

Chovestek symptom is shown in the remarkable increase in the mechanical excitability of the motor nerves. A slight tap for example in the course of the facial nerve will throw the muscles to which it is distributed into active contraction

Erb has shown that the electrical excitability of the nerves is also greatly increased, and Hoffmann has demonstrated the heightened excitability of the sensory nerves, the slightest pressure on which may cause paraesthesia in the parts to which it is distributed

Hoffmann's symptom

Oppenheim

Trol!

Gottstein reports a case (~~D. S. f. New Heil. 1865.~~) of relief of a long standing case of tetany by the use of the thyroid gland.

Bromide of potassium may be tried. In severe cases chloroform inhalations may be given. Massage, electricity, and the spinal ice-bag have also been used with success. Cases, however, may resist all treatment, and the spasms recur for many years.

XI. HYSTERIA.

Definition.—A state in which ideas control the body and produce morbid changes in its functions (Möbius).

Etiology.—The affection is most common in women, and usually appears first about the time of puberty, but the manifestations may continue until the menopause, or even until old age. Men and boys, however, are by no means exempt, and of late years hysteria in the male has attracted much attention. It occurs in all races, but is much more prevalent, particularly in its severer forms, in members of the Latin race. In this country the milder grades are common, but the graver forms are rare in comparison with the frequency with which they are seen in France.

Of predisposing causes, two are important—heredity and education. The former acts by endowing the child with a mobile, abnormally sensitive nervous organization. We see cases most frequently in families with marked neuropathic tendencies, the members of which have suffered from neuroses of various sorts. Education at home too often fails to inculcate habits of self-control. A child grows to girlhood with an entirely erroneous idea of her relations to others, and accustomed to have every whim gratified and abundant sympathy lavished on every woe, however trifling, she reaches womanhood with a moral organization unfitted to withstand the cares and worries of every-day life. At school, between the ages of twelve and fifteen, the most important period in her life, when the vital energies are absorbed in the rapid development of the body, she is often cramming for examinations and cooped in close school-rooms for six or eight hours daily. The result too frequently is an active, bright mind in an enfeebled body, ill adapted to subserve the functions for which it was framed, easily disordered, and prone to react abnormally to the ordinary stimuli of life. Among the more direct influences are emotions of various kinds, fright occasionally, more frequently love affairs, grief, and domestic worries. Physical causes less often bring on hysterical outbreaks, but they may follow directly upon an injury or develop during the convalescence from an acute illness or be associated with disease of the generative organs. The name *hysteria* indicates how important was believed to be the part played by the uterus in the causation of the disease. Opinions differ a good deal on this question, but undoubtedly in many cases there are ovarian and uterine disorders the rectification of which sometimes cures the disease. Sexual excess, particularly masturbation, is an important factor, both in girls and boys.

Symptoms.—A useful division is into the convulsive and non-convulsive varieties.

Convulsive Hysteria.—(*a*) *Minor Forms.*—The attack most commonly follows emotional disturbance. It may set in suddenly or be preceded by symptoms, called by the laity "hysterical," such as laughing and crying alternately, or a sensation of constriction in the neck, or of a ball rising in the throat—the *globus hystericus*. Sometimes, preceding the convulsive movements, there may be painful sensations arising from the pelvic, abdominal, or thoracic regions. From the description these sensations resemble auræ. They become more intense with the rising sensation of choking in the neck and difficulty in getting breath, and the patient falls into a more or less violent convulsion. It will be noticed that the fall is not sudden, as in epilepsy, but the subject falls, as a rule, easily, often picking a soft spot, like a sofa or an easy chair, and in the movements apparently exercises care to do herself no injury. Yet at the same time she appears to be quite unconscious. The movements are clonic and disorderly, consisting of to-and-fro motion of the trunk or pelvic muscles, and the head and arms are thrown about in an irregular manner. The paroxysm after a few minutes slowly subsides, then the patient becomes emotional, and gradually regains consciousness. When questioned the patient may confess to having some knowledge of the events which have taken place, but, as a rule, has no accurate recollection. During the attack the abdomen may be much distended with flatus, and subsequently a large amount of clear urine may be passed. These attacks vary greatly in character. There may be scarcely any movements of the limbs, but after a nerve storm the patient sinks into a torpid, semi-unconscious condition, from which she is roused with great difficulty. In some cases from this state the patient passes into a condition of catalepsy.

(*b*) *Major Forms; Hystero-epilepsy.*—This condition has been specially studied by Charcot and his pupils. Typical instances passing through the various phases are very rare in this country. The attack is initiated by certain prodromata, chiefly minor hysterical manifestations, either foolish or unseemly behavior, excitement, sometimes dyspeptic symptoms with tympanites, or frequent micturition. Areas of hyperæsthesia may at this time be marked, the so-called hysterogenic spots so elaborately described by Richet. These are usually symmetrical and situated over the upper dorsal vertebra, and in front in a series of symmetrically placed spots on the chest and abdomen, the most marked being those in the inguinal regions over the ovaries. Painful sensations or a feeling of oppression and a *globus* rising in the throat may be complained of prior to the onset of the convulsion, which, according to French writers, has four distinct stages: (1) Epileptoid condition, which closely simulates a true epileptic attack with tonic spasm (often leading to opisthotonos), grinding of the teeth, congestion of the face, followed by clonic convulsions, gradual relaxation, and coma. This attack lasts rather longer than a true epi-

leptic attack. (2) Succeeding this is a period which Charcot has termed *clownism*, in which there is an emotional display and a remarkable series of contortions or of cataleptic poses. (3) Then in typical cases there is a stage in which the patient assumes certain attitudes expressive of the various passions—ecstasy, fear, beatitude, or erotism. (4) Finally consciousness returns and the patient enters upon a stage in which she may display very varied symptoms, chiefly manifestations of a delirium with the most extraordinary hallucinations. Visions are seen, voices heard, and conversations held with imaginary persons. In this stage patients will relate with the utmost solemnity imaginary events, and make extraordinary and serious charges against individuals. This sometimes gives a grave aspect to these seizures, for not only will the patient at this stage make and believe the statements, but when recovery is complete the hallucination sometimes persists. We seldom see in this country attacks having this orderly sequence. Much more commonly the convulsions succeed each other at intervals for several days in succession. Here is a striking difference between hystero-epilepsy and true epilepsy. In the latter the status epilepticus, if persistent, is always serious, associated with fever, and frequently fatal, while in hystero-epilepsy attacks may recur for days without special danger to life. After an attack of hystero-epilepsy the patient may sink into a state of trance or lethargy, in which she may remain for days.

Non-convulsive Forms.—So complex and varied is the clinical picture of hysteria that various manifestations are best considered according to the systems which are involved.

(1) **Disorders of Motion.**—(*a*) *Paralyses.*—These may be hemiplegic, paraplegic, or monoplegic. Hysterical diplegia is extremely rare. The paralysis either sets in abruptly or gradually, and may take weeks to attain its full development. *There is no type or form of organic paralysis which may not be simulated in hysteria.* According to Weir Mitchell, the hemiplegias are most frequent in the ratio of four on the left to one on the right side. The face is not affected; the neck may be involved, but the leg suffers most. Sensation is either lessened or lost on the affected side. The hysterical paraplegia is more common than hemiplegia. The loss of power is not absolute; the legs can usually be moved, but do not support the patient. The reflexes may be increased, though the knee-jerk is often normal. A spurious ankle clonus may sometimes be present. The feet are usually extended and turned inward in the equino-varus position. The muscles do not waste and the electrical reactions are normal. Other manifestations, such as paralysis of the bladder or aphonia, are usually associated with the hysterical paraplegia. Hysterical monoplegias may be facial, crural, or brachial. A condition of ataxia sometimes occurs with paresis. The incoördination may be a marked feature, and there are usually sensory manifestations.

(*b*) *Contractures and Spasms.*—An extraordinary variety of spas-

modic affections occurs in hysteria, of which the most common are the following: The hysterical contractures may attack almost any group of voluntary muscles and be of the hemiplegic, paraplegic, or monoplegic type. They may come on suddenly or slowly, persist for months or years, and disappear rapidly. The contracture is most commonly seen in the arm, which is flexed at the elbow and wrist, and the fingers tightly grasp the thumb in the palm of the hand; more rarely the terminal phalanges are hyperextended as in athetosis. It may occur in one or in both legs, more commonly the former. The ankle clonus is present; the foot is inverted and the toes are strongly flexed. These cases may be mistaken for lateral sclerosis and the difficulty in diagnosis may really be very great. The spastic gait is very typical, and with the exaggerated knee-jerk and ankle clonus the picture may be characteristic. In 1879 I frequently showed such a case at the Montreal General Hospital as a typical example of lateral sclerosis. The condition persisted for more than eighteen months and then disappeared completely. Other forms of contracture may be in the muscles of the hip, shoulder, or neck; more rarely in those of the jaws —hysterical trismus—or in the tongue. Remarkable indeed are the local contractures in the diaphragm and abdominal muscles, producing a phantom tumor, in which just below and in the neighborhood of the umbilicus is a firm, apparently solid growth. According to Gowers, this is produced by relaxation of the recti and a spasmodic contraction of the diaphragm, together with inflation of the intestines with gas and an arching forward of the vertebral column. They are apt to occur in middle-aged women about the menopause, and are frequently associated with the symptoms of spurious pregnancy—*pseudo-cyesis*. The resemblance to a tumor may be striking, and I have known skilful diagnosticians to be deceived. The only safeguard is to be found in complete anæsthesia, when the tumor entirely disappears. Some years ago I went by chance into the operating-room of a hospital and found a patient on the table under chloroform and the surgeon prepared to perform ovariotomy. The tumor, however, had completely disappeared with full anæsthesia. Mitchell has reported an instance of a phantom tumor in the left pectoral region just above the breast, which was tender, hard, and dense.

Clonic spasms are more common in hysteria in this country than contractures. The following are the important forms: *Rhythmic hysterical spasm*. This, unfortunately, is sometimes known as rhythmic chorea or hysterical chorea. The movements may be of the arm, either flexion and extension, or, more rarely, pronation and supination. Clonic contractions of the sterno-cleido-mastoid or of the muscles of the jaws or of the rotatory muscles of the head may produce rhythmic movements of these parts. The spasm may be in one or both psoas muscles, lifting the leg in a rhythmic manner eight or ten times in a minute. In other instances the muscles of the trunk are affected, and every few moments there is a bowing movement—salaam convulsions—or the muscles of the back may

flexibilitas cerea

(If) attonitas and Catalepsy. Katatonia may be
features of hysteria (see Tuke's Dict. p. 724

In the type Reedii the reaction may or may not
persist during repose, but is increased or
provoked by voluntary movements
(see Hoyt's art. Am. Jr. 1853)

contract, causing strong arching of the vertebral column and retraction of the head. These movements may often alternate, as in a case in my wards, in which the patient on fine days had regular salaam convulsions, while on wet days the rhythmic spasm was in the muscles of the back and neck. Mitchell has described a rotatory spasm in which the patient rotated involuntarily, usually to the left. More unusual cases are those in which the contractions closely simulate paramyoclonus multiplex. A characteristic example of this was recently at my clinic. Hysterical athetosis is a rare form of spasm. *Tremor* may be a pure hysterical manifestation, occurring either alone or with paralysis and contracture. It most commonly involves the hands and arms; more rarely the head and legs. The movements are small and quick. Volitional or intentional tremor may exist, simulating closely the movements of insular sclerosis. Buzzard states that many instances of this disease in young girls are mistaken for hysteria.

(2) **Disorders of Sensation.**—*Anæsthesia* is most common, and usually confined to one half of the body. It may not be noticed by the patient. Usually it is accurately limited to the middle line and involves the mucous surfaces and deeper parts. The conjunctiva, however, is often spared. There may be hemianopia. This symptom may come on slowly or follow a convulsive attack. Sometimes the various sensations are dissociated and the anæsthesia may be only to pain and to touch. The skin of the affected side is usually pale and cool, and a pin-prick may not be followed by blood. With the loss of feeling there may be loss of muscular power. Curious trophic changes may be present, as in an interesting case of Weir Mitchell's, in which there was unilateral swelling of the hemiplegic side.

A phenomenon to which much attention has been paid is that of transference. By metallotherapy, the application of certain metals, the anæsthesia or analgesia can be transferred to the other side of the body. It has been shown, however, that this phenomenon may be caused by the electro-magnet and by wood and various other agents, and is probably entirely a mental effect. The subject has no practical importance, but it remains an interesting and instructive chapter in Gallic medical history.

Hyperæsthesia.—Increased sensitiveness and pains occur in various parts of the body. One of the most frequent complaints is of pain in the head, usually over the sagittal suture, less frequently in the occiput. This is described as agonizing, and is compared to the driving of a nail into the part; hence the name *clavus hystericus*. Neuralgias are common. Hyperæsthetic areas, the hysterogenic points, exist on the skin of the thorax and abdomen, pressure upon which may cause minor manifestations or even a convulsive attack. Increased sensitiveness exists in the ovarian region, but is not peculiar to hysteria. Pain in the back is an almost constant complaint of hysterical patients. The sensitiveness may be limited to certain spinous processes, or it may be diffuse. In hysterical women the pains in the abdomen may simulate those of gastralgia and of gastric

ulcer, or the condition may be almost identical with that of peritonitis; more rarely the abdominal pains closely resemble those of appendix disease.

Special Senses.—Disturbances of taste and smell are not uncommon and may cause a good deal of distress. Of ocular symptoms, retinal hyperæsthesia is the most common, and the patients always prefer to be in a darkened room. Retraction of the field of vision is common and usually follows a convulsive seizure. It may persist for years. The color perception may be normal even with complete anæsthesia, and in this country the achromatopsia does not seem to be nearly so common an hysterical manifestation as in Europe. Hysterical deafness may be complete and may alternate or come on at the same time with hysterical blindness.

(3) **Visceral Manifestations.**—*Respiratory Apparatus.*—Of disturbances in the respiratory rhythm, the most frequent, perhaps, is an exaggeration of the deeper breath, which is taken normally every fifth or sixth inspiration, or there may be a "catching" breathing, such as is seen when cold water is poured over a person. Hysterical dyspnœa is readily recognized, as there is no special distress and the pulse is usually normal. I have met with a remarkable case following trauma in which the respirations rose above one hundred and thirty in the minute. Among laryngeal manifestations aphonia is the most frequent and may persist for months or even years without other special symptoms of the disease. Spasm of the muscles may occur with violent inspiratory efforts and great distress, and may even lead to cyanosis. Hiccough, or sounds resembling it, may be present for weeks or months at a time. Among the most remarkable of the respiratory manifestations are the hysterical cries. These may mimic the sounds produced by animals, such as barking, mewing, or grunting, and in France epidemics of them have been repeatedly observed. Extraordinary cries may be produced, either inspiratory or expiratory. I saw at Wagner's clinic at Leipsic a girl of thirteen or fourteen, who had for many weeks given utterance to a remarkable inspiratory cry somewhat like the whoop of whooping-cough, but so intense that it was heard at a long distance. It was incessant, and the girl was worn to a skeleton. Attacks of gaping, yawning, and sneezing may also occur.

The hysterical cough is a frequent symptom, particularly in young girls. It may occur in paroxysms, but is often a dry, persistent, croaking cough, extremely monotonous and unpleasant to hear. Sir Andrew Clark has called attention to a loud, barking cough occurring about the time of puberty, chiefly in boys belonging to neurotic families. ~~The attacks, which last about a minute, recur frequently.~~

There is a peculiar form of hæmoptysis which may be very deceptive and lead to the diagnosis of pulmonary disorders. Wagner describes the sputum as a pale-red fluid—not so bright in color as in ordinary hæmoptysis, and on settling presents a reddish-brown sediment. It contains particles of food, pavement epithelium, red corpuscles, and micrococci, but

Hysterical amaurosis may occur in ~~boys~~ children. One must carefully distinguish between functional loss of power and simulation.

(9. 3. 94. Saw a boy with Berger who had received a blow on the rt. eye. ∴ black eye. evidently very severe. ∴ had headache ∴ after rt. well then for ∴ weeks but now for ∴ weeks has had defec vision. cannot read. no neurotic.

(Cyanobet hebetica)

† Hysterical bulimia. (Thayer case in a girl of 12.

Lasègue essay on Hyp Anorexia. Arch Gen de Med 1873, I

Mandeau Jr. de Med Chir et Phar. 1789. Juillet p 157

see Hazard. Am Jr. 1853. ii

no cylindrical or ciliated epithelium. It probably comes from the mouth or pharynx.

Digestive System.—Disturbed or depraved appetite, dyspepsia, and gastric pains are common in hysterical patients. The patient may have difficulty in swallowing the food, apparently from spasm of the gullet. There are instances in which the food seems to be expelled before it reaches the stomach. In other cases there is incessant gagging. In the hysterical vomiting the food is regurgitated without much effort and without nausea. This feature may persist for years without great disturbance of nutrition. The most striking and remarkable digestive disturbance in hysteria is the *anorexia nervosa* described by Sir William Gull. "To call it loss of appetite—anorexia—but feebly characterizes the symptom. It is rather an annihilation of appetite, so complete that it seems in some cases impossible ever to eat again. Out of it grows an antagonism to food which results at last and in its worst forms in spasm on the approach of food, and this in turn gives rise to some of those remarkable cases of survival for long periods without food" (Mitchell). As this goes on there may be an extreme degree of muscular restlessness, so that the patients wander about until exhausted. This feature has not been present in the cases which have come under my observation. Nothing more pitiable is to be seen in practice than an advanced case of this sort. It is usually in a young girl, sometimes as early as the eleventh or twelfth, more commonly between the fifteenth and twentieth years. The emaciation is frightful, and scarcely exceeded by that of cancer of the œsophagus. The patient finally takes to bed, and in extreme cases lies upon one side with the thighs and legs flexed, and contractures may occur. Food is either not taken at all or only upon urgent compulsion. The skin becomes wasted, dry, and covered with bran-like scales. No food may be taken for several weeks at a time, and attempts to feed may be followed by severe spasms. Although the condition looks so alarming, these cases, when removed from their home surroundings and treated by Weir Mitchell's method, sometimes recover in a remarkable way. Death, however, may follow with extreme emaciation. In a fatal case recently under my care the girl weighed only forty-nine pounds. No lesions were found post mortem.

Among intestinal symptoms flatulency is one of the most distressing, and is usually associated with the condition of peristaltic unrest (Küssmaul). Frequent discharges of fæces may be due to disturbance in either the small or large bowel. An obstinate form of diarrhœa is found in some hysterical patients, which proves very intractable and is associated especially with the taking of food. It seems an aggravated form of the looseness of bowels to which so many nervous people are subject on emotion or the tendency which some have to diarrhœa immediately after eating. An entirely different form is that produced by what Mitchell calls the irritable rectum, in which scybala are passed frequently during the day, sometimes with great violence. Constipation is more frequent, however, and may be

due to a loss of power in the muscles of the bowel or in the abdominal muscles. In extreme cases the bowels may not be moved for two or three weeks, leading to great accumulation of fæces. Other disturbances are ano-spasm or intense pain in the rectum apart from any fissure.

Cardio-vascular.—Rapid action of the heart on the slightest emotion, with or without the subjective sensation of palpitation, is often a source of great distress. A slow pulse is less frequent. Pains about the heart may simulate angina, the so-called hysterical or pseudo-angina, which has already been considered. Flushes in various parts are among the most common symptoms, and may be seen in the head, back, hands, or feet. Sweating occasionally occurs.

Among the more remarkable vaso-motor phenomena are the so-called stigmata or hæmorrhages in the skin, such as were present in the celebrated case of Louise Lateau. In many cases these are undoubtedly fraudulent, but if, as appears credible, such bleeding may exist in the hypnotic trance, there seems no reason to doubt its occurrence in the trance of prolonged religious ecstasy.

Joint Affections.—To Sir Benjamin Brodie and Sir James Paget we owe the recognition of these extraordinary manifestations of hysteria. Perhaps no single affection has brought more discredit upon the profession, for the cases are very refractory, and finally fall into the hands of a charlatan or faith-healer, under whose touch the disease may disappear at once. Usually it affects the knee or the hip, and may follow a trifling injury. The joint is usually fixed, sensitive, and swollen. The surface may be cool, but sometimes the local temperature is increased. To the touch it is very sensitive and movement causes great pain. In protracted cases the muscles about the joint are somewhat wasted, and in consequence it looks larger. The pains are often nocturnal, at which time the local temperature may be much increased. While, as a rule, neuromimetic joints yield to proper management, there are interesting instances in the literature in which organic change has succeeded the functional disturbance. In the remarkable case reported in Weir Mitchell's lectures, the hysterical features were pronounced, and, on account of the chronicity, the disease of the knee-joint was considered organic by such an authority as Billroth. Sands operated and found the joint surfaces normal, and the thickening to be due to non-tuberculous inflammatory products outside the capsule.

Mental Symptoms.—The psychical condition of an hysterical patient is always abnormal, and the disease occupies the ill-defined territory between sanity and insanity. In a large number of cases the patients are really insane, particularly in the perversion witnessed in the moral sphere. Not the slightest dependence can be placed upon their statements, and they will for months or years deceive friends, relatives, and physician. This appears to result partly, but not wholly, from a morbid craving for sympathy. It is really due to an entire unhinging of the moral nature.

Hysterical bladder.

Hysterical breast.

Intermittent hysterical may see Rev. Neurology
~~result of~~ manifestation of hysteria
~~at~~ occurring ~~paroxysms~~ only in the knee
or the jaw or in several joints, sometimes
with transient paresis

Status hysterici

I have been accustomed to speak of this condition
as the status hysterici

Hysterical patients may become insane and display persistent hallucinations and delirium, alternating perhaps with emotional outbursts of an aggravated character. For weeks or months they may be confined to bed, entirely oblivious to their surroundings, with a delirium which may simulate that of delirium tremens, particularly in being associated with loathsome and unpleasant animals. The nutrition may be maintained, but in these cases there is always a very heavy, foul breath. With seclusion and care recovery usually takes place within three or four months. At the onset of these attacks and during convalescence the patients must be incessantly watched, as a suicidal tendency is by no means uncommon.

Of hysterical manifestations in the higher centres that of trance is the most remarkable. This may develop spontaneously without any convulsive seizure, but more frequently, in this country at least, it follows hysteroid attacks. Catalepsy, a condition in which the limbs are plastic and remain in any position in which they are placed, may or may not be present with this condition.

The Metabolism in Hysteria.—The studies of Gilles de la Tourette and Cathelineau, under Charcot's direction, have shown that in the ordinary forms of hysteria the urine does not show quantitative or qualitative changes, but in the severe types, characterized by convulsions, etc., there are important modifications: reduction in the urates and phosphates; the ratio of the earthy to the alkaline phosphates, normally 1 : 3, is 1 : 2, or even 1 : 1. The urine is also reduced in amount. They think that these changes might sometimes serve to differentiate convulsive hysteria from epilepsy, in which there is always an increase in the solid constituents after a seizure.

Hysterical Fever.—In hysteria the temperature, as a rule, is normal. The cases with fever may be grouped as follows: (*a*) Instances in which the fever is the sole manifestation. These are rare, but I have seen at least two cases in which the chronic course, the retention of the nutrition, and the entirely negative condition of the organs left no other diagnosis possible. In a case recently under observation the patient has had for four or five years an afternoon rise of temperature, reaching usually to 102° or 103°. She was well nourished and presented no pronounced hysterical symptoms, but there was a marked neurotic history on one side and a form of interrupted sighing respiration so often seen in hysteria.

(*b*) Cases of hysterical fever with spurious local manifestations. These are very troublesome and deceptive cases. The patient may be suddenly taken ill with pain in various regions and elevation of temperature. The case may simulate meningitis. There may be pain in the head, vomiting, contracted pupils, and retraction of the neck—symptoms which may persist for weeks—and some anomalous manifestation during convalescence may alone indicate to the physician that he has had to deal with a case of hysteria, and has not, as he perhaps flattered himself, cured a case of meningitis. Mary Putnam Jacobi, in a recent article on hysterical fever,

mentions a case in the service of Cornil which was admitted with dyspnœa, slight cyanosis, and a temperature at 39° C. The condition proved to be hysterical. There is also an hysterical pseudo-phthisis with pain in the chest, slight fever, and the expectoration of a blood-stained mucus. The cases of hysterical peritonitis may also show fever. Only by incessant watchfulness can mistakes be prevented in these cases.

(*c*) *Hysterical Hyperpyrexia.*—It is a suggestive fact that the cases of paradoxical temperatures reported of late years, in which the thermometer has registered 112° to 120° or more, have been in women. Fraud has been practised in some of these, but in others the high fever has been associated with neurotic features and may really have been of an hysterical character.

Diagnosis.—Inquiry into the occurrence of previous manifestations and the mental conditions may give important information. These questions, as a rule, should not be asked the mother, who of all others is least likely to give satisfactory information about the patient's condition. The occurrence of the globus hystericus, of emotional attacks, of weeping and crying, are always suggestive. The points of difference between the convulsive attacks and true epilepsy were referred to in their description, and as a rule little difficulty is experienced in distinguishing between the two conditions. The hysterical paralyses are very variable and apt to be associated with anæsthesia. The contractures may at times be very deceptive, but the occurrence of areas of anæsthesia, of retraction of the visual field, and the development of minor hysterical manifestations, give valuable indications. The contractures disappear under full anæsthesia. Special care must be taken not to confound the spastic paraplegia of hysteria with lateral sclerosis.

The visceral manifestations are usually recognized without much difficulty. The practitioner has constantly to bear in mind the strong tendency in hysterical patients to practise deception.

Treatment.—The prophylaxis in hysteria may be gathered from the remarks on the relation of education to the disease. The successful treatment of hysteria demands qualities possessed by few physicians. The first element is a due appreciation of the nature of the disease on the part of the physician and friends. It is pitiable to think of the misery which has been inflicted on these unhappy victims by the harsh and unjust treatment which has resulted from false views of the nature of the trouble; on the other hand, worry and ill-health, often the wrecking of mind, body, and estate, are entailed upon the near relatives in the nursing of a protracted case of hysteria. The minor manifestations, attacks of the vapors, the crying and weeping spells, are not of much moment and rarely require treatment. The physical condition should be carefully looked into and the mode of life regulated so as to insure system and order in everything. A congenial occupation offers the best remedy for many of these manifestations. Any functional disturbance should be at-

have to be accepted, though their explanation is impossible under our present laws. Jacobi has recently reported a case in which the temperature rose to 146°.7 (63°.5 C.). The Omaha case in which the temperature was recorded at 170°.4 has, I am assured on good authority, proved to be a fraud.

tended to and a course of tonics prescribed. Special attention should be paid to the action of the bowels.

Valerian and asafœtida are often of service. For the pains in various parts, particularly in the back, the thermo-cautery and static electricity will be found invaluable. Morphia should be withheld. In the convulsive seizures, particularly in the minor forms, it is often best, after settling the patient comfortably, to leave her. When she comes to, and finds herself alone and without sympathy, the attacks are less likely to be repeated. There is, as a rule, no cure for the hysterical manifestations of women, otherwise in good health, who are, as Mitchell says, "fat and ruddy, with sound organs and good appetites, but ever complain of pains and aches, and ever liable on the least emotional disturbance to exhibit a quaint variety of hysterical phenomena."

To treat hysteria as a physical disorder is, after all, radically wrong. It is essentially a mental and emotional anomaly, and the important element in the treatment is moral control. At home, surrounded by loving relatives who misinterpret entirely the symptoms and have no appreciation of the nature of the disease, the severer forms of hysteria can rarely be cured. The necessary control is impossible; hence the special value of the method introduced by Weir Mitchell, which is particularly applicable to the advanced cases which have become chronic and bedridden. The treatment consists in isolation, rest, diet, massage, and electricity. Separation from friends and sympathetic relatives must be absolute, and can rarely, if ever, be obtained in the individual's home. An essential element in the treatment is an intelligent nurse. No small share of the success which has attended the author of this plan has been due to the fact that he has persistently chosen as his allies bright, intelligent women. The details of the plan are as follows: The patient is confined to bed and not allowed to get up, nor, at first, in aggravated cases, to read, write, or even to feed herself. Massage is used daily, at first for twenty minutes or half an hour, subsequently for a longer period. It is essential as a substitute for exercise. The induction current is applied to the various muscles and to the spine. Its use, however, is not so essential as that of massage. The diet may at first be entirely of milk, four ounces every two hours. It is better to give skimmed milk, and it may be diluted with soda water or barley water and, if necessary, peptonized. After a week or ten days the diet may be increased, the amount of milk still being kept up. A chop may be given at midday, a cup of coffee or cocoa with toast or bread and butter or a biscuit with the milk. The patients usually fatten rapidly as the solid food is added, and with the gain there is, as a rule, a diminution or cessation of the nervous symptoms. The milk is the essential element in the diet, and is itself amply sufficient.

The remarkable results obtained by this method are now universally recognized. The plan is more applicable to the lean than to fat, flabby hysterical patients. Not only is it suitable for the more obstinate varie-

ties of hysteria with bodily manifestations, but in the cases with mental symptoms the seclusion and separation from relatives and friends are particularly advantageous. In the hysterical vomiting Debove's method of forced feeding may be used with benefit. For the innumerable minor manifestations of hysteria and for the simulations the indications for treatment are usually clear. Of late, hypnotism has been extensively used in the treatment of hysteria. Occasionally in cases of hysterical contractions or paralysis it is of benefit, but any one who has seen the development of this method as practised at present in France must feel that it is a two-edged sword and that the constant repetition in the same patient is fraught with danger. In the cases which we have tried here the success has not been marked.

XII. NEURASTHENIA.

Definition.—A condition of weakness or exhaustion of the nervous system.

The term, invented by Beard, covers an ill-defined, motley group of symptoms, which may be either general and the expression of derangement of the entire system, or local, limited to certain organs; hence the terms cerebral, spinal, cardiac, and gastric neurasthenia. In certain respects it is the physical counterpart of insanity. As the essential feature in the latter condition is the abnormal response to stimuli, from within or without, upon the higher centres presiding over the mind, so neurasthenia appears to be the expression of a morbid, unhealthy reaction to stimuli acting on the nervous centres which preside over the functions of organic life. No hard and fast line can be drawn between neurasthenia and certain mental states, particularly hysteria and hypochondria.

Etiology.—Although the causes are apparently varied, they may be grouped as hereditary and acquired.

(*a*) *Hereditary.*—We do not all start in life with the same amount of nerve capital. Parents who have been the subjects of nervous complaints or of mental troubles transmit to their children an organization which is defective in what, for want of a better term, we must call "nerve force." Such individuals start handicapped, and furnish a considerable proportion of our neurasthenic patients. So long as they are content to transact a moderate business with their life capital, all may go well, but there is no reserve, and in the emergencies which constantly arise in the exigencies of modern life these small capitalists go under and come to us as bankrupts.

(*b*) *Acquired.*—The functions, though perverted most readily in persons who have inherited a feeble organization, may also be damaged by exercise which is excessive in proportion to the strength—i. e., by strain. The cares and anxieties attendant upon the gaining of a livelihood may be borne without distress, but in many persons the strain becomes excess-

ive and is first manifested as *worry*. The individual loses the distinction between essentials and non-essentials, trifles cause annoyance, and the entire organism reacts with unnecessary readiness to slight stimuli, and is in a state which the older writers called irritable weakness. If such a condition be taken early and the patient given rest, the balance is quickly restored. In this group may be placed a large proportion of the neurasthenics which we see in this country, particularly among business men. Other causes more subtle, yet potent, and less easily dealt with, are the worries attendant upon love affairs, religious doubts, and the sexual passion.

Symptoms.—These are extremely varied, and may be general or localized; more often a combination of both. The appearance of the patient is suggestive, sometimes characteristic, but difficult to describe. Loss of weight and slight anæmia may be present. The physical debility may reach a high grade and the patient may be confined to bed. Mentally the patients are usually low-spirited and despondent, in women frequently emotional.

The local symptoms may dominate the situation, in which case the clinical picture is of the so-called cerebral or spinal neurasthenia. Other local forms are cardio-vascular, gastric, and sexual.

In the cerebral form the symptoms are chiefly connected with an inability to perform the ordinary mental work. Thus a row of figures cannot be correctly added, the dictation or the writing of a few letters is a source of the greatest worry, the transaction of petty details in business is a painful effort, and there is loss of power of fixed attention. With this condition there may be no headache, the appetite may be good, and the patient may sleep well. As a rule, however, there are sensations of fulness and weight or flushes, if not actual headache. Sleeplessness is a frequent concomitant, and may be the first manifestation. Some of these patients are good-tempered and cheerful, but a majority are moody, irritable, and depressed.* The special senses may be disturbed, particularly vision. An aching or weariness of the eyeballs after reading a few minutes or flashes of light are common symptoms. A difference between the pupils may be present.

When the *spinal symptoms* predominate—spinal irritation or spinal neurasthenia—in addition to many of the features just mentioned, the patients complain of weariness on the least exertion, of weakness, pain in the back, and of aching pains in the legs. There may be spots of local tenderness on the spine. Occasionally there may be disturbances of sensation, particularly a feeling of numbness and tingling, and the reflexes may be increased. The aching pain in the back or in the back of the neck is the most constant complaint in these cases. In women it is often

* For an exhaustive consideration of the mental symptoms of neurasthenia, see the Shattuck Lecture, by Cowles. Boston Medical and Surgical Journal, 1891.

impossible to say whether this condition is one of neurasthenia or hysteria.

In other cases the *cardio-vascular* symptoms are the most distressing, and may occur with only slight disturbance of the cerebro-spinal functions, though the conditions may be combined. Palpitation of the heart, irregular and very rapid action, and pains in the cardiac region are the most common symptoms. The slightest excitement may be followed by increased action of the heart, and the patients frequently have the idea that they suffer from serious disease of this organ.

Vaso-motor disturbances constitute a special feature of many cases. Flushes of heat and transient hyperæmia of the skin may be very distressing symptoms. Profuse sweating may occur, either local or general, and sometimes nocturnal. The pulse may show interesting features, owing to the extreme relaxation of the peripheral arterioles. The arterial throbbing may be everywhere visible, almost as much as in aortic insufficiency. The pulse, too, may under these circumstances have a somewhat water-hammer quality. The capillary pulse may be seen in the nails, on the lips, or on the margins of a line drawn upon the forehead, and I have on several occasions seen pulsation in the veins of the back of the hand. A characteristic symptom in some cases is the *throbbing aorta*. The epigastric pulsation may be extremely forcible and suggest the existence of abdominal aneurism. The subjective sensations associated with it may be very unpleasant, particularly when the stomach is empty.

The general features of gastro-intestinal neurasthenia have been dealt with under the section of nervous dyspepsia. The connection of these cases with dilatation of the stomach, floating kidney, and the condition which Glénard calls *enteroptosis* has already been mentioned.

Sexual neurasthenia is a condition in which there is an irritable weakness of the sexual organs manifested by nocturnal emissions, unusual depression after intercourse, and often by a distressing dread of impotence. The mental condition of these patients is most pitiable, and they fall an easy prey to quacks and charlatans of all kinds.

In all forms of neurasthenia the condition of the urine is important. Many cases are complicated with the symptoms of the condition known as lithæmia, and so marked may this be that some have indeed made a special form of lithæmic neurasthenia. Polyuria may be present, but is more common in hysteria. With disturbed digestion the urates and oxalates may be in excess.

The *diagnosis* is readily made. It is sometimes difficult to distinguish the cases from hysteria, and this is not surprising, as we cannot always differentiate the two conditions. Neurasthenia occurs chiefly in men; in fact, it is in many ways in them the equivalent of hysteria.

1 See also Burns "On the Heart" 1809 p. 267 et seq. on Asthma

This "
causes of premature and unnatural exhaustion in
organism." as Allan Burns calls it,

XIII. THE TRAUMATIC NEUROSES

(*Railway Brain and Railway Spine; Traumatic Hysteria*).

Definition.—A morbid condition following shock which presents the symptoms of neurasthenia or hysteria or of both. The condition is known as "railway brain" and "railway spine."

Erichsen regarded the condition as the result of inflammation of the meninges and cord, and gave it the name railway spine. Walton and J. J. Putnam, of Boston, were the first to recognize the hysterical nature of many of the cases,* and to Westphal's pupils we owe the name traumatic neurosis.

Etiology.—The condition follows an accident, often in a railway train, in which injury has been sustained, or succeeds a shock or concussion, from which the patient may apparently not have suffered in his body. A man may appear perfectly well for several days, or even a week or more, and then develop marked symptoms of the neurosis. Bodily shock or concussion is not necessary. The affection may follow a profound mental impression; thus, an engine driver ran over a child, and received thereby a very severe shock, subsequent to which the most pronounced symptoms of neurasthenia developed. Severe mental strain combined with bodily exposure may cause it, as in a case of a naval officer who was wrecked in a violent storm and exposed for more than a day in the rigging before he was rescued. A slight blow, a fall from a carriage or on the stairs may suffice.

Symptoms.—The cases may be divided into three groups: simple neurasthenia, cases with marked hysterical manifestations, and cases with severe symptoms indicating or simulating organic disease.

(*a*) *Simple Traumatic Neurasthenia.*—The first symptoms usually develop a few weeks after the accident, which may or may not have been associated with an actual trauma. The patient complains of headache and tired feelings. He is sleepless and finds himself unable to concentrate his attention properly upon his work. A condition of nervous irritability develops, which may have a host of trivial manifestations, and the entire mental attitude of the person may for a time be changed. He dwells constantly upon his condition, gets very despondent and low-spirited, and in extreme cases melancholia may develop. He may complain of numbness and tingling in the extremities, and in some cases of much pain in the back. The bodily functions may be well performed, though such patients usually have, for a time at least, disturbed digestion and loss in weight. The physical examination may be entirely negative. The reflexes are slightly increased, as in ordinary neurasthenia. The pupils may be unequal; the cardio-vascular changes already described in neurasthenia may be present in a marked degree. According as the symptoms are more

* See La Neurasthénie, par L. Bouveret, Paris, 1891.

spinal or more cerebral, the condition is known as railway brain or railway spine.

(2) *Cases with Marked Hysterical Features.*—Following an injury of any sort, neurasthenic symptoms, like those described above, may develop, and in addition symptoms regarded as characteristic of hysteria. The emotional element is prominent, and there is but slight control over the feelings. The patients have headache, backache, and vertigo. A violent tremor may be present, and indeed constitutes the most striking feature of the case. I have recently seen an engineer who developed subsequent to an accident a series of nervous phenomena, but the most marked feature was an excessive tremor of the entire body, which was specially manifest during emotional excitement. The most pronounced hysterical symptoms are the sensory disturbances. As first noted by Putnam and Walton, hemianæsthesia may occur as a sequence of traumatism. This is a common symptom in France, but rare in England and in this country. In a considerable number of cases of traumatic neuroses which I have seen only one presented typical hemianæsthesia. A second, more common, manifestation is limitation of the field of vision.

Remarkable disturbances may develop in some of these cases. A few months ago I saw a man who had been struck by an electric car, whose chief symptom was an extraordinary increase in the number of respirations. He was a stout, powerfully built man, and presented practically no other symptom than dyspnœa of the most extreme grade. At the time of observation his respirations were over 130 per minute, and he stated that they had been counted at over 150.

(3) *Cases in which the Symptoms suggest Organic Disease of the Brain and Cord.*—As a result of spinal concussion, without fracture or external injury, there may subsequently develop symptoms suggestive of organic disease, which may come on rapidly or at a late date. In a case reported by Leyden the symptoms following the concussion were at first slight and the patient was regarded as a simulator, but finally the condition became aggravated and death resulted. The post-mortem showed a chronic pachymeningitis, which had doubtless resulted from the accident. The cases in this group about which there is so much discussion are those which display marked sensory and motor changes. Following an accident in which the patient has not received external injury a condition of excitement may develop within a week or ten days; he complains of headache and backache, and on examination sensory disturbances are found, either hemianæsthesia or areas on the skin in which the sensation is much benumbed; or painful and tactile impressions may be distinctly felt in certain regions, and the temperature sense is absent. The distribution may be bilateral and symmetrical in limited regions or hemiplegic in type. Limitation of the field of vision is usually marked in these cases, and there may be disturbance of the senses of taste and smell. The superficial reflexes may be diminished; usually the deep reflexes are exaggerated. The

achromatopsia may exist on the anaesthetic side

pupils may be unequal; the motor disturbances are variable. The French writers describe cases of monoplegia with or without contracture, symptoms upon which Charcot lays great stress as a manifestation of profound hysteria. The combination of sensory disturbances—anæsthesia or hyperæsthesia—with paralysis, particularly if monoplegic, and the occurrence of contractures without atrophy and with normal electrical reactions, may be regarded as distinctive of hysteria.

In rare cases following trauma and succeeding to symptoms which may have been regarded as neurasthenic or hysterical, there are organic changes which may prove fatal. That this sequence occurs is demonstrated clearly by recent post-mortem examinations. The features upon which the greatest reliance can be placed as indicating ~~definite~~ organic change are optic atrophy, bladder symptoms, particularly in combination with tremor, paresis, and exaggerated reflexes.

The anatomical changes in this condition have not been very definite. When death follows spinal concussion within a few days there may be no apparent lesion, but in some instances the brain or cord has shown punctiform hæmorrhages. Edes has reported four cases in which a gradual degeneration in the pyramidal tracts followed concussion or injury of the spine; but in all these cases there was marked tremor and the spinal symptoms developed early or followed immediately upon the accident. Post-mortems upon cases in which organic lesions have supervened upon a traumatic neurosis are extremely rare. Bernhardt reports an instance of a man, aged thirty-three, who in 1886 received a kick from a horse on the epigastrium and subsequently developed the symptom-complex of neurasthenia and hysteria with attacks of vertigo and great psychical depression. He afterward had more marked mental symptoms and attacks of unconsciousness. He committed suicide and the brain and cord showed a beginning multiple sclerosis in the white matter, which was possibly associated with an advanced grade of arterio-sclerosis. In a second case a man, aged forty-two, received a shock in a railway accident in July, 1884. He was rendered unconscious and had a slight injury in the buttock region. In a few weeks symptoms of traumatic neurosis developed, particularly great depression of spirits, with headache and sensory disturbances in the feet and hands. Tremor and great weakness were complained of when he attempted to work. There was no increase in the reflexes. The case was regarded as an instance of simulation and a defect in objective symptoms favored this view. Subsequently this judgment was reversed, but he did not improve. He died in January, 1889, with symptoms of cardiac dyspnœa. Macroscopically the brain and cord appeared normal. There was extreme arterio-sclerosis, particularly of the vessels of the brain and cord. In the latter there were scattered areas of degeneration in the white substance, and degeneration in the sympathetic ganglia.

I have entered somewhat fully into this question because of its extreme

importance and on account of the paucity of the observations upon cases which have subsequently developed symptoms of organic disease. Examples of it are extremely rare. So far as I know no case with autopsy has been reported in this country, nor have I seen an instance in which the clinical features pointed to an organic disease which had followed upon a traumatic neurosis.

Diagnosis.—A condition of fright and excitement following an accident may persist for days or even weeks, and then gradually pass away. The symptoms of neurasthenia or of hysteria which subsequently develop present nothing peculiar and are identical with those which occur under other circumstances. Care must be taken to avoid simulation, and, as in these cases the condition is largely subjective, this is sometimes extremely difficult. In a careful examination a simulator will often reveal himself by exaggeration of certain symptoms, particularly sensitiveness of the spine, and by increasing voluntarily the reflexes. It may require a careful study of the case to determine whether the individual is honestly suffering from the symptoms of which he complains. A still more important question in these cases is, Has the patient organic disease? The symptoms given under the first two groups of cases may exist in a marked degree and may persist for several years without the slightest evidence of organic change. It must be noted that in the two autopsies above referred to the patients were the subjects of extreme arterio-sclerosis, with which, in all probability, the areas of multiple sclerosis were associated. Hemianæsthesia, limitation of the field of vision, monoplegia with contracture, may all be present as hysterical manifestations, from which recovery may be complete. In our present knowledge the diagnosis of an organic lesion should be limited to those cases in which optic atrophy, bladder troubles, and signs of sclerosis of the cord are well marked—indications either of degeneration of the lateral columns or of multiple sclerosis.

Prognosis.—A majority of patients with traumatic hysteria recover. In railway cases, so long as litigation is pending and the patient is in the hands of lawyers the symptoms usually persist. Settlement is often the starting point of a speedy and perfect recovery. I have known return to health after the persistence of the most aggravated symptoms with complete disability of from three to five years' duration. On the other hand, there are a few cases in which the symptoms persist even after the litigation has been closed; the patient goes from bad to worse and psychoses develop, such as melancholia, dementia, or occasionally progressive paresis. And, lastly, in extremely rare cases, organic lesions may develop as a sequence of the traumatic neurosis.

The function of the physician acting as medical expert in these cases consists in determining (*a*) the existence of actual disease, and (*b*) its character, whether simple neurasthenia, severe hysteria, or an organic lesion. The outlook for ultimate recovery is good except in cases which present the more serious symptoms above mentioned. Nevertheless, it must be borne

These are cases in which the organic and functional features are curiously blended. Dr. J. G. Sawtelle of the 2d U. S. Cav. a man aged about 30 was in March last struck on the back of the neck with a side of beef."

He gradually developed right hemianesthesia complete & analgesia hemianopsia, hemiamblyopsia in the right eye (could not tell words at all) & gradually began to drag the left leg: Curious jerky hemiplegic gait. Great increase in reflexes Hand?

Lately all attacks like petit mal.

What is the lesion, if any?

in mind that traumatic hysteria is one of the most intractable affections which we are called upon to treat.

Treatment of Neurasthenia.—Many patients come under our care a generation too late for satisfactory treatment, and it may be impossible to restore the exhausted capital. In other instances, the recovery takes place rapidly, the patient remains well for a few months or a year, and then overwork, or even the ordinary wear and tear of life, again prostrates him. Other persons drift into a condition of chronic invalidism or become slaves to morphia or chloral. In the case of business or professional men, in whom the condition develops as a result of overwork or overstudy, it may be sufficient to enjoin absolute rest with change of scene and diet. A trip abroad, with a residence for a month or two in Switzerland, or, if there are symptoms of nervous dyspepsia, a residence at one of the Spas, will usually prove sufficient. The excitement of the large cities abroad should be avoided. Better still for these cases, if they carry it out, is a life in the woods or on the plains. Three months of tent-life in the Adirondacks or the same length of time in the Rocky Mountains will sometimes cure the most marked cases of this kind. Such a plan is not, however, within the circumstances of all. In a much larger class, including a large proportion of neurasthenic women, a systematic Weir Mitchell treatment rigidly carried out should be tried (see hysteria). For obstinate and protracted cases, particularly if combined with the chloral or morphia habit, no other plan is so satisfactory. The treatment of the gastric and intestinal symptoms so important in this condition has already been considered. In milder grades of the condition massage alone will be found very useful. For the irregular pains, particularly in the back and neck, the thermo-cautery is invaluable. Medicines are of little avail. Strychnia in full doses is often beneficial. For the relief of sleeplessness all possible measures should be resorted to before the employment of drugs.

XIV. OTHER FORMS OF FUNCTIONAL PARALYSIS.

I. Periodical Paralysis.

I have already referred to the remarkable periodical paralysis of the ocular muscles, which may recur at intervals for many years. There is a form of periodical paralysis involving the general muscles, which may recur with great regularity, and which is also a "family" affection. In Westphal's case, a boy of twelve, the attacks began in the eighth year, and at first recurred every four or six weeks, and lasted from a few hours to two days. Goldflam* has described a family in which twelve members were affected with this disease, the heredity being through the mother.

* Zeitschrift für klinische Medicin, Bd. xix, 1891.

Cousot has also met with a family in which the mother and four children were attacked. The disease occurs in youth, and the tendency to the attacks diminishes with age.

The clinical picture is very much alike in all the recorded cases. The paralysis involves, as a rule, the arms and legs. It comes on when the patients are in full health, and without any apparent cause, often during sleep. Sometimes it begins with weakness in the limbs, a sensation of weariness and sleepiness, not often with sensory symptoms. The paralysis is usually complete within the first twenty-four hours, beginning in the legs, to which in rare instances it is confined. The muscles of the neck are sometimes involved, and occasionally those of the tongue and pharynx. The cerebral nerves and the special senses are, as a rule, uninvolved. The attacks are afebrile, sometimes with low temperatures and slow pulse. The deep reflexes are reduced, sometimes abolished, and the skin reflexes may be feeble. One of the most remarkable features is the extraordinary reduction or complete abolition of the faradic excitability, both of muscles and of nerves.

Improvement begins sometimes in the course of a few hours or after a day or two, and the paralysis disappears completely, and the patient is perfectly well. As mentioned, the attacks may recur every few weeks, in some instances even daily; more commonly, an interval of one or two weeks elapses between the attacks. Goldflam suggests that the paralysis is due to an auto-intoxication, and that the poisonous material acts upon the nerve-endings in the muscles. He has made experiments with the urine of a case which showed that during the attacks the toxic properties of this secretion were materially increased. From the recurring, periodic character of the attacks they have been supposed to be due to malaria, but of this there is no evidence.

II. Astasia; Abasia.

These terms, indicating respectively inability to stand and inability to walk, have been applied by Charcot and Blocq to diseased conditions characterized by loss of the power of standing or of walking with retention of muscular power, coördination, and sensation. Blocq's definition is as follows: "A morbid state in which the impossibility of standing erect and walking normally is in contrast with the integrity of sensation, of muscular strength, and of the coördination of the other movements of the lower extremities." The condition forms a symptom group, not a morbid entity, and is probably a functional neurosis. Knapp in a recent paper analyzes the 50 cases reported in the literature. Twenty-five of these were in men, 25 in women. In 21 cases hysteria was present; in 3, chorea; in 2, epilepsy; and in 4, intention psychoses. As a rule, the patients, though able to move the feet and legs perfectly when in bed, are either unable to walk properly or cannot stand at all. The disturbances have been very varied,

Narcolepsy –

and different forms have been recognized. The commonest, according to Knapp's analysis of the recorded cases, is the paralytic, in which the legs give out as the patient attempts to walk and "bend under him as if made of cotton." "There is no rigidity, no spasm, no incoördination. In bed, sitting, or even while suspended, the muscular strength is found to be good." Other cases are associated with spasm or ataxia; thus there may be movements which stiffen the legs and give to the gait a somewhat spastic character. In other instances there are sudden flexions of the legs, or even of the arms, or a saltatory, spring-like spasm. In a majority of the cases it is a manifestation of a neurosis allied to hysteria.

The cases, as a rule, recover, particularly in young persons. Relapses are not uncommon. The rest treatment and static electricity should be employed.

V. VASO-MOTOR AND TROPHIC DISORDERS.

I. RAYNAUD'S DISEASE.

Definition.—A vascular disorder, probably dependent upon vaso-motor influences, characterized by three grades of intensity: (*a*) Local syncope, (*b*) local asphyxia, and (*c*) local or symmetrical gangrene.

Local Syncope.—This condition is seen most frequently in the extremities, producing the condition known as dead fingers or dead toes. It is analogous to that produced by great cold. The entire hand may be affected with the fingers; more commonly only one or more of the fingers. This feature of the disease rarely occurs alone, but is generally associated with local asphyxia. The common sequence is as follows: On exposure to slight cold or in consequence of some emotional disturbance the fingers become white and cold, or both fingers and toes are affected. The pallor may continue for an indefinite time, though usually not more than an hour or so; then gradually a reaction follows and the fingers get burning hot and red. This does not necessarily occur in all the fingers together; one finger may be as white as marble, while the adjacent ones are of a deep red or plum color.

Local Asphyxia.—Chilblains form the mildest grade of this condition. It usually follows the local syncope, but it may come on independently. The fingers and toes are oftenest affected, next in order the ears; more rarely portions of the skin on the arms and legs. During an attack the fingers alone, sometimes the hands, also swell and become intensely congested. In the most extreme grade the fingers are perfectly livid, and the capillary circulation is almost stagnant. The swelling causes stiffness and usually pain, not acute, but due to the tension and distention of the skin. Sometimes there is marked anæsthesia. Attacks of this sort

may recur for years, and be brought on by the slightest exposure to cold or in consequence of disturbances, either mental or, in some instances, gastric. Apart from this unpleasant symptom the general health may be very good. The attacks may recur only at long intervals or during the winter time.

Local or Symmetrical Gangrene.—The mildest grade of this condition follows the local asphyxia, in the chronic cases of which small necrotic areas are sometimes seen at the tips of the fingers. Sometimes the pads of the fingers and of the toes are quite cicatricial from repeated slight losses of this kind. So also when the ears are affected there may be superficial loss of substance at the edge. The severer cases, which terminate in extensive gangrene, are fortunately rare.

In an attack the local asphyxia persists in the fingers. The terminal phalanges, or perhaps only one finger, become black, cold, and insensible. The skin begins to necrose and superficial gangrenous blebs appear. Gradually a line of demarkation shows itself and a portion of one or more of the fingers sloughs away. The resulting loss of substance is much less than the appearance of the hand or foot would indicate, and a condition which looks as if the patient would lose all the fingers or half of a foot may result perhaps in only a slight superficial loss in the phalanges. In severer cases the greater portion of a finger or the tip of the nose may be lost. Occasionally the disease is not confined to the extremities, but affects symmetrical patches on the limbs or trunk, and may pass on to rapid gangrene. These severe types of cases occur particularly in young children, and death may result within three or four days. The attacks are usually very painful, and the motion of the part is much impaired. In some cases numbness and tingling persist for a long time.

There are remarkable concomitant symptoms in Raynaud's disease to which a good deal of attention has been paid of late years. Hæmoglobinuria may develop during an attack, or may take the place of an outbreak. In such instances the affection is usually brought on by cold weather. In a case reported by H. M. Thomas from my clinic, Raynaud's disease occurred for three successive winters and always in association with hæmoglobinuria. The attacks were sometimes preceded by a chill. Several cases of the kind are found in Barlow's appendix to his translation of Raynaud's paper for the New Sydenham Society. The onset with a chill, as in the case just mentioned, has doubtless given rise to the idea that the disease is in some way associated with ague. Cerebral symptoms, particularly mental torpor and transient loss of consciousness, have also been noticed ~~in some cases~~. The case just mentioned with hæmoglobinuria had epilepsy with the attacks. Exposure on a cold day would bring on an epileptic seizure with the local asphyxia and bloody urine. Occasionally joint affections develop, particularly anchylosis and thickening of the phalangeal articulations. Southey has reported a case in which mania developed, and Barlow an instance in which the woman had delusions. Peripheral neuritis has been found in several cases.

The pathology of this remarkable disease is still obscure. Raynaud suggested that the local syncope was produced by contraction of the vessels, which seems likely. The asphyxia is dependent upon dilatation of the capillaries and small veins, probably with the persistence of some degree of spasm of the smaller arteries. There are two totally different forms of congestion, which may be shown in adjacent fingers; one may be swollen, of a vivid red color, extremely hot, the capillaries and all the vessels fully distended, and the anæmia produced by pressure may be instantaneously obliterated; the adjacent finger may be equally swollen, absolutely cyanotic, stone cold, and the anæmia produced by pressure takes a long time to disappear. In the latter case the arterioles are probably still in a condition of spasm.

Treatment.—In many cases the attacks recur for years unaffected by treatment. Mild attacks require no treatment. In the severer forms of local asphyxia, if in the feet, the patient should be kept in bed with the legs elevated. The toes should be wrapped in cotton-wool. The pain is often very intense and may require morphia. Carefully applied, systematic massage of the extremities is sometimes of benefit. Galvanism may be tried. Barlow advises immersing the affected limb in salt water and placing one electrode over the spine and the other in the water.

X

II. ANGIO-NEUROTIC OEDEMA.

Definition.—An affection characterised by the occurrence of local oedematous swellings, more or less limited in extent, and of transient duration. Severe colic is sometimes associated with the outbreak. There is a marked hereditary disposition in the disease. The affection has been specially studied by Quincke, Jamieson, J. E. Graham, and Matas.

Symptoms.—The oedema appears suddenly and is usually circumscribed. It may appear in the face; the eyelid is a common situation; or it may involve the lips or cheek. The backs of the hands, the legs, or the throat may be attacked. Usually the condition is transient, associated perhaps with slight gastro-intestinal disturbance, and the affection is of little moment. There may be premonitory symptoms of the outbreak of the oedema, such as tingling, heat, redness, etc. In some instances, previous to the outbreak of the oedema, sudden oedema of the larynx may show itself. Two members of the family just referred to died of this complication, in one member of this family whom I saw recently in attacks the swell-

Another patient the subject has had for years attacks of Raynaud's disease, has had many attacks of transient hemiplegia on one side or the other, then on the right side with aphasia

Milroy of Omaha has ~~reported~~ described a form of hereditary oedema ~~founding~~ in 22 cases in six generations in which in the affected individuals there resulted from birth an oedema only of one or both legs without any special inconvenience or any increase in the mischief.

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ings came on in different parts; for example, the under lip would be swollen to such a degree that the mouth could not be opened. The hands enlarge suddenly, so that the fingers cannot be bent. The attacks recur every three or four weeks. Accompanying them are usually gastro-intestinal attacks, severe colic, pain, nausea, and sometimes vomiting. The colic is of great intensity and usually requires morphia. Arthritis apparently does not occur.

The disease has affinities with urticaria, the giant form of which is probably the same disease. There is a form of severe purpura, often with urticarial manifestations, which is also associated with marked gastro-intestinal crises. Quincke regards the condition as a vaso-motor neurosis, under the influence of which the permeability of the vessels is suddenly increased.

The *treatment* is very unsatisfactory. In the cases associated with anæmia and general nervousness, tonics, particularly large doses of strychnia, do good; but too often the disease resists all treatment.

III. FACIAL HEMI-ATROPHY.

An affection characterized by progressive wasting of the bones and soft tissues of one side of the face. The atrophy begins, as a rule, in childhood, but in a few cases has not come on until middle age. It begins diffusely, but in some instances has started at one spot on the skin and has gradually spread, involving at first the subcutaneous tissues, then the muscles and the bones, more particularly the upper jaw. The wasting is sharply limited at the middle line, and the appearance of the patient is very remarkable, the face looking as if made up of two halves from different persons. There is usually change in the color of the skin and the hair falls. Owing to the wasting of the alveolar processes the teeth become loose and ultimately fall out. The wasting involves the tissues of the orbit, and the eye on the affected side is sunken. In a majority of the cases the atrophy has been confined to one side of the face, but there are instances on record in which the disease was bilateral, and a few cases in which there were areas of atrophy on the back and on the arm of the same side. The disease is rare. Sachs has collected 97 cases from the literature.

Two autopsies have been made. In Mendel's case there was the terminal stage of an interstitial neuritis in all the branches of the trigeminus, from its origin to the periphery, most marked in the superior maxillary branch.

In Homén's case, which came on rapidly and scarcely belongs to the typical form of the disease, a tumor was found pressing upon the Gasserian ganglion and the trigeminus nerve.

The disease is recognized at a glance. The facial asymmetry asso-

Milroy of Omaha, has described cases of a form of hereditary œdema, 22 individuals in 24 generations, in which there existed from birth a solid œdema of one or both legs, without any special inconvenience or any progressive increase of the disease

~~and allied affections~~

Many cases have now been reported.

Somnolence is ~~noted in all of Cohen's cases~~ has been noted in many cases, and in one case of Packard's.

ciated with congenital wryneck must not be confounded with progressive facial hemi-atrophy. The precise nature of the disease is still doubtful.

IV. ACROMEGALIA.

Definition.—A dystrophy characterized by abnormal processes of growth, chiefly in the bones of the face and extremities.

The term was introduced by Marie, and signifies large extremities.

Etiology.—~~Nothing definite is known concerning the cause of the disease.~~ It occurs rather more frequently in women. Of the 38 cases analyzed in the monograph of Souza-Leite, 16 were in men and 22 in women. The disease usually begins about the twenty-fifth year, though in some instances as late as the fortieth. Rheumatism, syphilis, and the specific fevers have preceded the development of the disease, but probably have no special connection with it. In this country ~~five or six cases have~~ been reported, two by J. E. Graham, of Toronto.

Symptoms.—In a well-marked case the disease presents most characteristic features. The hands and feet are greatly enlarged, but are not deformed, and can be used freely. The hypertrophy is general, involving all the tissues, and gives a curious spade-like character to the hands. The wrists may be enlarged, but the arms are rarely affected. The feet are involved like the hands and are uniformly enlarged. The big toe may be much larger in proportion. The nails are usually broad and large. The head increases in volume, but not as much in proportion as the face, which becomes much elongated and enlarged in consequence of the increase in the size of the superior and inferior maxillary bones. The latter in particular increases greatly in size, and often projects below the upper jaw. The alveolar processes are widened and the teeth separated. The soft parts also increase in size, and the nostrils are large and broad. The eyelids are sometimes greatly thickened, and the ears enormously hypertrophied. The tongue in some instances becomes greatly enlarged. Late in the disease the spine may be affected and the back bowed—kyphosis. The bones of the thorax may slowly and progressively enlarge. With this gradual increase in size the skin of the hands and face may appear normal. Sometimes it is slightly altered in color, coarse, or flabby, but it has not the dry, harsh appearance of the skin in myxœdema. The muscles are sometimes wasted. Changes in the thyroid have been found, but are not constant. The gland has been normal in some, atrophied in others, and in a third group of cases enlarged. Erb, who has made an elaborate study of the disease, has noticed an area of dulness over the manubrium sterni, which he thought possibly due to the persistence or enlargement of the thymus. Headache is not uncommon. Menstrual disturbance may occur early, and there may be suppression. In some instances vision has been involved, owing to a gradual atrophy of

the optic nerve. The disease may persist for fifteen, twenty, or more years.

The *pathological anatomy* has been studied in a few cases. In addition to enlargement of the bones, which is a true hypertrophy, enormous enlargement of the hypophysis (pituitary body) has been found, and some have regarded the disease as associated in some way with this. Less constant have been the changes in the thymus and in the thyroid. In some instances the peripheral nerves have been involved. The most exhaustive anatomical study made as yet is that published by Arnold, of Heidelberg, on the case which was described clinically by Friedreich and Erb.

As stated, the true nature of the disease is unknown. Marie regards it as a systemic dystrophy, analogous to myxœdema and possibly due to the morbid condition of the pituitary body, just as myxœdema is associated with disease of the thyroid.

Diagnosis.—The disease must be carefully separated from the *osteitis deformans* of Paget, in which the shafts of the long bones are chiefly involved, and in the head the bones of the cranium, but not those of the face. Marie states that in Paget's disease the face is triangular with the base upward; in acromegalia it is ovoid, or egg-shaped, with the large end downward; while in myxœdema it is round and full-moon shaped. The disease must not be confounded with the instances of congenital or progressive hypertrophy of a single member, as of the leg or arm, the so-called giant growth, in which the various proportions are maintained.

Lastly, Marie has separated from acromegalia a group of cases characterized by hypertrophy of the bones of the extremities and of the shafts, producing great disability. The spine is also affected and curvature takes place. The fingers are characteristic. The terminal phalanges become bulbous, enlarged, and the nails are curved, which gives the appearance of the so-called Hippocratic finger, a very different condition indeed from the flattened terminal phalanges of acromegalia. Etiologically, Marie regards this form as associated in some way with pulmonary troubles. Thus, for instance, two of the patients had purulent pleurisy, the cases of Ewald and of Saundby had new growths in the lungs, and others presented chronic bronchitis. Marie, therefore, terms this form *osteo-arthropathie pneumique*. It is doubtful, however, as Arnold states in his exhaustive study of Friedreich's case, whether this form can really be separated from acromegalia.

The treatment does not appear to have any influence upon the progress of the disease.

Here may be mentioned a remarkable dystrophy, met with so far only in women, known as *sclerodactyle*, in which there are symmetrical involvements of the fingers, which become deformed, shortened, and atrophied. The skin becomes thickened, of a waxy color, and is sometimes pigmented. Bullæ and ulcerations have been met with in some instances, and a great deformity of the nails. The disease has usually followed exposure, and the

To go on p. 994. before author

Syphilis also

The hypoph-

A lung extract has been used ~~with~~ in some cases of osteo-arthropathie pneumique.

9.12.94 I saw remarkable case today in Sir B Richardson wards in the Temperance Hospital: all fingers involved. nevus in one, sclerodema

patients are much worse during the winter and are curiously sensitive to cold. There may be changes in the skin of the feet, but the deformity similar to that which occurs in the hand has not been noted. Some of the cases have presented in addition diffuse sclerodermatous changes of the skin of other parts. An admirable description of the disease has been given by Gordinier.*

V. SCLERODERMA.

Definition.—A condition of localized or diffuse induration of the skin.

Two forms are recognized, the localized or circumscribed, which corresponds to the keloid of Addison and to morphea, and the diffuse, in which large areas are involved.

In the *circumscribed form* there are patches, ranging from a few centimetres in diameter to the size of the hand or larger, in which the skin has a waxy or dead-white appearance and to the touch is brawny, hard, and inelastic. Sometimes there is a preliminary hyperæmia of the skin, and subsequently there are changes in color, either areas of pigmentation or of complete atrophy of the pigment—leucoderma. The sensory changes are rarely marked. The secretion of sweat is diminished or entirely abolished. The disease is more common in women than in men, and is situated most frequently about the breasts and neck, sometimes in the course of the nerves. The patches may develop with great rapidity, and may persist for months or years; sometimes they disappear in a few weeks.

The *diffuse form*, though less common, is more serious. It develops first in the extremities or in the face, and the patient notices that the skin is unusually hard and firm, or that there is a sense of stiffness or tension in making accustomed movements. Gradually a diffuse, brawny induration develops and the skin becomes firm and hard, and so united to the subcutaneous tissues that it cannot be picked up or pinched. The skin may look natural, but more commonly is glossy, drier than normal, and unusually smooth. Of 44 cases, in 24 the first appearances were on the arms, in 7 on the legs, in 1 on both, in 10 on the face and neck, and in 2 on the trunk (Dinkler). The disease may gradually extend and involve the skin of an entire limb; in rare cases, it becomes universal, the face is expressionless, the lips cannot be moved, mastication is impossible, and it becomes extremely difficult to feed the patient. The hands become fixed, the fingers immobile, on account of the extreme induration of the skin over the joints. The disease is chronic, lasting for many months or many years. There are instances on record of its persistence for more than twenty years. Recovery may occur, or the disease may be arrested.

* American Journal of the Medical Sciences, January, 1889.

The patients are apt to succumb to pulmonary complaints or to nephritis. Rheumatic troubles have been noticed in some instances; in others, endocarditis. The pathology of the disease is unknown. It is usually regarded as a tropho-neurosis, probably dependent upon changes in the arteries of the skin leading to connective-tissue overgrowth.

The patients require to be warmly clad and to be guarded against exposure, as they are particularly sensitive to changes in the weather. Frictions with oil, and galvanism are recommended.

AINHUM.

Here a brief reference may be made to the remarkable trophic lesion described by Da Silva Lima, which is met with in negroes in Brazil, Africa, India, and occasionally in the Southern States. It is confined to the toes, usually the little toe, and begins as a furrow on the line of the digito-plantar fold. This gradually deepens, the end of the toe enlarges, and, usually without inflammation or pain, the toe falls off. The process may last some years. Cases have been reported in this country by Hornaday, Pittman, F. J. Shepherd, and Morrison.

A combination of Sclerodema and Reynaud disease may be associated as in two cases described by Stephen Mackenzie. I have seen an instance in which the primary symptoms were those of local asphyxia of the fingers and in which with extensive sclerodema of the arms & hands, & face there were cyanosis and swelling of the skin of the feet without any coronary indication.

SECTION IX.

DISEASES OF THE MUSCLES.

I. MYOSITIS.

Definition.—Inflammation of the voluntary muscles.

A primary myositis occurs as an acute or subacute affection, and is probably dependent on some unknown infectious agent. Several characteristic cases have been described of late years. The case of E. Wagner may be taken as a typical example. A tuberculous but well-built woman entered the hospital, complaining of stiffness in the shoulders and a slight œdema of the back of the hands and forearms. There was paræsthesia, the arms became swollen, the skin tense, and the muscles felt doughy. Gradually the thighs became affected. The disease lasted about three months. The post-mortem showed slight pulmonary tuberculosis; all the muscles except the glutei, the calf, and abdominal muscles were stiff and firm, but fragile, and there were serous infiltration, great proliferation of the interstitial tissue, and fatty degeneration. Similar cases have been reported by Unverricht, Hepp, and Jacoby of New York. In the case reported by Jacoby the muscles were firm, hard, and tender, and there was slight œdema of the skin The duration of the cases is usually from one to three months, though there are instances in which it has been longer The swelling and tenderness of the muscles, the œdema, and the pain naturally suggest trichinosis, and indeed Hepp speaks of it as a pseudo-trichinosis. The nature of the disease is unknown. Senator's case presented marked disorders of sensation, and there is a question whether the peripheral nerves are not involved with the muscles. Wagner suggests that some of these cases were examples of acute progressive muscular atrophy. The separation from trichinosis can be made only by removing a portion of the muscle. There are septic cases in which a diffuse, purulent infiltration of the muscles of different regions occurs. Instances have been reported in which this has been described as the primary affection, the condition of the muscles even passing on to gangrene.

A remarkable affection is *myositis ossificans progressiva*, in which portions of the muscles undergo a progressive calcification.

II. IDIOPATHIC MUSCULAR ATROPHY

(Primary Muscular Dystrophy—Erb).

Definition.—Muscular wasting, with or without an initial hypertrophy, beginning in various groups of muscles, usually progressive in character, and dependent on primary changes in the muscles themselves. A marked hereditary disposition is met with in the disease.

Before considering the primary muscular atrophies it may be well to summarize briefly the chief conditions under which muscular atrophy occurs. These are:

(1) Acute or chronic lesions of the nuclei of the motor path, which may be (*a*) cortical, as a direct result of a cerebral lesion; (*b*) medullary, as in chronic bulbar paralysis; (*c*) spinal, either acute, as in poliomyelitis of children, or chronic, as in the progressive muscular atrophy of the simple or of the spastic type.

(2) Neuritic muscular atrophy, following a local neuritis due to trauma, a multiple neuritis due to alcohol, lead, and the infectious diseases. In this same category probably may be placed the muscular atrophies associated with joint-disease, the progressive hemi-atrophy of the face, and the atrophy sometimes found in cases of hysteria.

(3) Conditions of the muscles themselves—primary muscular atrophy.

Etiology.—The most important factor is heredity. Many members of the same family may be attacked through several generations. Males, as a rule, are more frequently affected than females. The disease is usually transmitted through the mother, though she may not herself be the subject. As many as twenty or thirty cases have been described in five generations. Isolated cases, however, are not uncommon. The disease usually sets in before puberty, but may be as late as the twentieth or twenty-fifth year, or in some instances even later. No etiological factors of any moment are known other than heredity.

Clinical Forms.—Two chief types may be recognized: (1) With primary hypertrophy, the pseudo-hypertrophic muscular paralysis; and (2) with primary atrophy.

Pseudo-hypertrophic Muscular Paralysis.—The first symptom noticed is, as a rule, clumsiness in the movements of the child, and on examination certain muscles or groups of muscles seem to be enlarged, particularly those of the calves. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps, and infraspinatus, are the next most frequently involved, and may stand out with great prominence. The muscles of the neck, face, and forearm rarely suffer. Sometimes only a portion of a muscle is involved. With this hypertrophy of some muscles there is wasting of others, particularly the lower portion of the pectorals and the latissimus dorsi. The attitude when standing is very characteristic. The legs are far apart, the shoulders thrown back, the spine is greatly curved, and the abdomen protrudes. The gait is waddling and awkward. In

getting up from the floor the position assumed, as so well known now through Gowers's figures, is pathognomonic. The patient first turns over in the all-fours position and raises the trunk with his arms; the hands are then moved along the ground until the knees are reached; then with one hand upon a knee he lifts himself up, grasps the other knee, and gradually pushes himself into the erect posture, as it has been expressed, by climbing up his legs. The striking contrast between the feebleness of the child and the powerful-looking pseudo-hypertrophic muscles is very characteristic.

The course of the disease is slow, but progressive. Wasting proceeds and finally all traces of the enlarged condition of the muscles disappears. At this late period distortions and contractions are common.

Primary Atrophic Form.—Here, too, there is the same marked tendency to involvement of different members of a family. Five or six different types have been described, but it seems more rational to group them together under the designation of idiopathic muscular atrophy. In all of the cases the atrophy begins, as a rule, before the twentieth year. According to the site of the primary atrophy different forms have been described. In the *juvenile type* of Erb the affection begins about the fifteenth or the twentieth year and involves the muscles of the upper arm and shoulder and the gluteal and thigh muscles. In the facio-scapulo-humeral type of Landouzy and Déjérine the muscles of the face are early involved with those of scapulo-humeral groups. This form occurs usually in families, and the onset may be delayed until the twentieth or thirtieth year. Leyden describes an *hereditary form*, beginning in the lower extremities and back, which may be associated with hypertrophy of the calves. Another type has been described by Charcot and Tooth—the *peroneal form;* but there is still some doubt whether this is not in reality a myelopathy and more closely related to chronic polio-myelitis anterior. In this form the atrophy begins in the muscles of the legs, usually in the extensors of the great toe, and afterward in the common extensors and the peroneal groups. The cases usually begin early, and the heredity through the mother has been traced in several remarkable series, particularly that of Herringham's. Fibrillary contractions and the reaction of degeneration are present. Nerve degeneration has been found in the peripheral parts, and ascending degeneration of the columns of Goll.

Morbid Anatomy.—The spinal cord and peripheral nerves have been found normal in cases of pseudo-hypertrophic muscular paralysis and in the forms of idiopathic muscular atrophy. The muscles in the pseudo-hypertrophic condition present great variations in the size of the muscle fibres, some of which may be hypertrophied and others wasted. In the early stage the hypertrophy of the fibres may be very pronounced and the nuclei of the sarcolemma are greatly increased. In some instances, too, the fibres have been fissured longitudinally. At a later stage the muscular

fibres are wasted and largely replaced by connective tissue and fat. In the primary atrophic form wasting of the fibres, increase in the interstitial tissue, and the development of fat are the most marked features. Except in the peroneal type, about which there is still doubt, no affection of the nerves or cord has been determined.

Diagnosis.—The primary myopathies can usually be readily distinguished from the cerebral, myelopathic, and neuritic forms.

(*a*) In the cerebral atrophy loss of power usually precedes the atrophy, which is either of a monoplegic or hemiplegic type.

(*b*) In the myelopathic or spinal muscular atrophy the distinctions are clearly marked. *Polio-myelitis anterior chronica* begins in the small muscles of the hand, a situation rarely if ever affected by the primary myopathies, which involve first those of the calves, the trunk, the face, or the shoulder-girdle. In the myelopathic atrophy the reaction of degeneration is present and fibrillary twitchings occur in both the atrophied and non-atrophied muscles. In many cases in addition to the wasting in the arms there is a spastic condition in the legs and increase in the reflexes. The myelopathic atrophies come on late in life; the myopathic forms develop, as a rule, early. In the primary muscular atrophies heredity plays an important *rôle*, which in the myelopathic is quite subsidiary.

(*c*) In the neuritic muscular atrophies, whether due to lead or to trauma, the general characters and the mode of onset are distinctive. In the cases of multiple neuritis seen for the first time at a period when the wasting is marked there is often difficulty, but the absence of family history and the distribution are important features. Moreover, the paralysis is out of proportion to the atrophy. Sensory symptoms may be present, and in the cases in which the legs are chiefly involved there is usually the *steppage* gait so characteristic of peripheral neuritis.

The outlook in the primary myopathies is bad. The wasting progresses uniformly, uninfluenced by treatment. Erb holds that by electricity and massage the progress is occasionally arrested. The general health should be carefully looked after, moderate exercise allowed, frictions with oil applied to the muscles, and when the patient becomes bedfast, as is inevitable sooner or later, care should be taken to prevent contractures in awkward positions.

III. THOMSEN'S DISEASE; MYOTONIA CONGENITA.

Definition.—An hereditary disease characterized by tonic cramp of the muscles on attempting voluntary movements. The disease received its name from the physician who first described it, in whose family it has existed for five generations.

Etiology.—All the typical cases have occurred in family groups; a few isolated instances have been described in which similar symptoms

have been present. The disease is rare in this country and in England; it seems more common in Germany and in Scandinavia.

Symptoms.—The disease comes on in childhood. It is noticed that on account of the stiffness the children are not able to take part in ordinary games. The peculiarity is noticed only during voluntary movements. The contraction which the patient wills is slowly accomplished; the relaxation which the patient wills is also slow. The contraction often persists for a little time after he has dropped an object which he has picked up. In walking, the start is difficult; one leg is put forward slowly, it halts from stiffness for a second or two, and then after a few steps the legs become limber and he walks without any difficulty. The muscles of the arms and legs are those usually implicated; rarely facial, ocular, or laryngeal muscles. Emotion and cold aggravate the condition. In some instances there is mental weakness. The sensation and the reflexes are normal. The condition of the muscles is interesting. The patients appear well and are muscular, and there is sometimes a definite hypertrophy of the muscles. The force is scarcely proportionate to the size. Erb has described a characteristic reaction of the nerve and muscle to the electrical currents—the so-called myotonic reaction, the chief feature of which is that normally the contractions caused by either constant stream (the [illegible] slowly and relax slowly, and vermicular, wave-like contractions pass from the cathode to the anode.

The disease is incurable, but it may be arrested temporarily. The nature of the affection is unknown. There is an extraordinary increase in the size of the voluntary fibres. According to Hale White, who has recently treated the subject in an admirable and critical manner, the fibres may be more than double the width of those of the normal muscles. The nuclei and the interstitial tissue may be increased and some of the fibres contain vacuoles. No post-mortem has been made. No treatment for the condition is known.

IV. PARAMYOCLONUS MULTIPLEX.

An affection, described by Friedrich, characterised by clonic contractions, chiefly of the muscles of the extremities, occurring either constantly or in paroxysms.

The cases have usually been in males and the disease has followed emotional disturbance, fright, or straining. The contractions are usually bilateral and may vary from fifty to one hundred and fifty in the minute. Occasionally tonic spasms occur. It is not accompanied by any sensory or motor disturbances. In the intervals between the attacks there may be tremors of the muscles. In the severe spasms the movements may be very

* Only Reported Before 1880.

In the only autopsy made
Déjerine and Sottas have ~~reported one autopsy~~ found
hypertrophy of the "~~fasciculi primitivi~~" with
multiplication of the nuclei of all the muscles, including
the diaphragm but not the heart. The spinal cord &
the nerves were intact

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* Guy's Hospital Reports, 1889.

violent; the body is tossed about, and it is sometimes difficult to keep the patient in bed. In a case which I saw at the Bicêtre the patient was perfectly quiet so long as his legs were tied down with a sheet, but as soon as this was removed the clonic spasms occurred in the legs and muscles of the back and tossed the body about in the bed from side to side. The patient uttered a curious expiratory grunt. The nature of the disease is unknown.

SECTION X.

THE INTOXICATIONS, SUN-STROKE, OBESITY.

I. ALCOHOLISM.

SECTION X.

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I. ALCOHOLISM.

(1) **Acute Alcoholism.**—When a large quantity of alcohol is taken, its influence on the nervous system is manifested in muscular incoördination, mental disturbance, and, finally, narcosis. The individual presents a flushed, sometimes slightly cyanosed face, a full pulse, with deep but rarely stertorous respirations. The pupils are dilated. The temperature is frequently below normal, particularly if the patient has been exposed to cold. Perhaps the lowest reported temperatures have been in cases of this sort. An instance is on record in which the patient on admission to hospital had a temperature of 24° C. (ca. 75° F.), and ten hours later the temperature had not risen to 91°. The unconsciousness is rarely so deep that the patient cannot be roused to some extent, and in reply to questions he mutters incoherently. Muscular twitchings may occur, but rarely convulsions. The breath has a heavy alcoholic odor.

The diagnosis is not difficult, yet mistakes are frequently made. Persons are sometimes brought to hospital by the police supposed to be drunk when in reality they are dying from apoplexy. Too great care cannot be exercised, and the patient should receive the benefit of the doubt. In some instances the mistake has arisen from the fact that a person who has been drinking heavily has been stricken with apoplexy. In this condition the coma is usually deeper, stertor is present, and there may be evidence of hemiplegia in the greater flaccidity of the limbs on one side. The subject has already been considered in the section upon uræmic coma.

(2) **Chronic Alcoholism.**—In moderation, wine, beer, and spirits may be taken throughout a long life without impairing the general health.

According to Payne, the poisonous effects of alcohol are manifested (1) as a functional poison, as in acute narcosis; (2) as a tissue poison, in which its effects are seen on the parenchymatous elements, particularly epithelium and nerve, producing a slow degeneration, and on the blood-vessels, causing thickening and ultimately fibroid changes; and (3) as a checker

of tissue oxidation, since the alcohol is consumed in place of the fat. This leads to fatty changes and sometimes to a condition of general steatosis.

The chief effects of chronic alcohol poisoning may be thus summarized:

Nervous System.—Functional disturbance is common.—Unsteadiness of the muscles in performing any action is a constant feature. The tremor is best seen in the hands and in the tongue. The mental processes may be dull, particularly in the early morning hours, and the patient is unable to transact any business until he has had his accustomed stimulant. Irritability of temper, forgetfulness, and a change in the moral character of the individual gradually come on. The judgment is seriously impaired, the will enfeebled, and in the final stages dementia may supervene. The relation of chronic alcoholism to insanity has been much discussed. According to Savage, of 4,000 patients admitted to the Bethlehem Hospital, 133 gave drink as the cause of their insanity. Chronic alcoholism is believed by many to be one of the special causes of dementia paralytica, but the opinions of experts on this question are still discordant. Savage states that not more than seven per cent are caused by alcohol alone. In many cases it is certainly one of the important elements in the strain which leads to this breakdown.

No characteristic changes are found in the nervous system. Hæmorrhagic pachymeningitis is not very uncommon. Opacity and thickening of the pia-arachnoid membranes, with more or less wasting of the convolutions, generally occur. These are in no way peculiar to chronic alcoholism, but are found in old persons and in chronic wasting diseases. In the very protracted cases there may be chronic encephalo-meningitis with adhesions of the membranes. By far the most striking effect of alcohol on the nervous system is the production of the alcoholic neuritis, which has already been considered.

Digestive System.—Catarrh of the stomach is the most common symptom. The toper has a furred tongue, heavy breath, and in the morning a sensation of sinking at the stomach until he has his dram. The appetite is usually impaired and the bowels are constipated. These features are associated with a chronic catarrh of the stomach.

Alcohol produces definite changes on the liver, leading to the various forms of cirrhosis already described. The effect is probably a primary degenerative change in the liver-cells, although many good observers still hold that the poison acts first upon the connective-tissue elements. It is probable that a special vulnerability of the liver-cells is necessary in the etiology of alcoholic cirrhosis. There are cases in which comparatively moderate drinking for a few years has been followed by cirrhosis; on the other hand, the livers of persons who have been steady drinkers for thirty or forty years may show only a moderate grade of sclerosis. With the gastric and hepatic disorders the facies often becomes very characteristic. The venules of the cheeks and nose are dilated; the latter becomes enlarged,

red, and may present the condition known as *acne rosacea*. The eyes are watery, the conjunctivæ hyperæmic and sometimes bile-tinged.

Kidneys.—The influence of chronic alcoholism upon these organs is by no means so marked. According to Dickinson the total of renal disease is not greater in the drinking class, and he holds that the effect of alcohol on the kidneys has been much overrated. Formad has directed attention to the fact that in a large proportion of chronic alcoholics the kidneys are increased in size. The Guy's Hospital statistics support this statement, and Pitt notes that in forty-three per cent of the bodies of hard drinkers the kidneys were hypertrophied without showing morbid change. The typical granular kidney seems to result indirectly from alcohol through the arterial changes.

It was formerly thought that alcohol was in some way antagonistic to tuberculous disease, but the observations of late years indicate clearly that the reverse is the case and that chronic drinkers are much more liable to both acute and pulmonary tuberculosis. It is probably altogether a question of altered tissue-soil, the alcohol lowering the vitality and enabling the bacilli more readily to develop and grow.

(3) **Delirium Tremens** (*mania a potu*) is really only an incident in the history of chronic alcoholism, and results from the long-continued action of the poison on the brain. The condition was first accurately described early in this century by Sutton, of Greenwich, who had numerous opportunities for studying the different forms among the sailors. One of the most thorough and careful studies of the disease was made by Ware, of Boston. A spree in a temperate person, no matter how prolonged, is rarely if ever followed by delirium tremens; but in the case of an habitual drinker a temporary excess is apt to bring on an attack. It sometimes develops in consequence of the sudden withdrawal of the alcohol. There are circumstances which in a heavy drinker determine, sometimes with abruptness, the onset of delirium. Such are an accident, a sudden fright or shock, and an acute inflammation, particularly pneumonia. At the outset of the attack the patient is restless and depressed and sleeps badly, symptoms which cause him to take alcohol more freely. After a day or two the characteristic delirium sets in. The patient talks constantly and incoherently; he is incessantly in motion, and desires to go out and attend to some imaginary business. Hallucinations of sight and hearing develop. He sees objects in the room, such as rats, mice, or snakes, and fancies that they are crawling over his body. The terror inspired by these imaginary objects is great, and has given the popular name "horrors" to the disease. The patients need to be watched constantly, for in their delusions they may jump out of the window or escape. Auditory hallucinations are not so common, but the patient may complain of hearing the roar of animals or the threats of imaginary enemies. There is much muscular tremor; the tongue is covered with a thick white fur, and when protruded is tremulous. The pulse is soft, rapid, and readily compressed. There is usually

fever, but the temperature rarely registers above 102° or 103°. In fatal cases it may be higher. Insomnia is a constant feature. On the third or fourth day in favorable cases the restlessness abates, the patient sleeps, and improvement gradually sets in. The tremor persists for some days, the hallucinations gradually disappear, and the appetite returns. In more serious cases the insomnia persists, the delirium is incessant, the pulse becomes more frequent and feeble, the tongue dry, the prostration extreme, and death takes place from gradual heart-failure.

Diagnosis.—The clinical picture of the disease can scarcely be confounded with any other. Cases with fever, however, may be mistaken for meningitis. By far the most common error is to overlook some local disease, such as pneumonia or erysipelas, or an accident, as a fractured rib, which in a chronic drinker may precipitate an attack of delirium tremens. In every instance a careful examination should be made, particularly of the lungs. It is to be remembered that in the severer forms, particularly the febrile cases, congestion of the bases of the lungs is by no means uncommon. Another point to be borne in mind is the fact that pneumonia of the apex is apt to be accompanied by delirium similar to *mania a potu*.

Prognosis.—Recovery takes place in a large proportion of the cases in private practice. In hospital practice, particularly in the large city hospitals to which the debilitated patients are taken, the death rate is higher. Gerhard states that of 1,241 cases admitted to the Philadelphia Hospital 121 proved fatal. Recurrence is frequent, almost indeed the rule, if the drinking is kept up.

Treatment.—Acute alcoholism rarely requires any special measures, as the patient sleeps off the effects of the debauch. In the case of profound alcoholic coma it may be advisable to wash out the stomach, and if collapse symptoms occur the limbs should be rubbed and hot applications made to the body. Should convulsions supervene, chloroform may be carefully administered. In the acute, violent alcoholic mania the hypodermic injection of apomorphia, one eighth or one sixth of a grain, is usually very effectual, causing nausea and vomiting, and rapid disappearance of the maniacal symptoms.

Chronic alcoholism is a condition very difficult to treat, and once fully established the habit is rarely abandoned. The most obstinate cases are are those with marked hereditary tendency. Withdrawal of the alcohol is the first essential. This is most effectually accomplished by placing the patient in an institution, in which he can be carefully watched during the trying period of the first week or ten days of abstention. The absence of temptation in institution life is of special advantage. For the sleeplessness the bromides or hyoscine may be employed. Quinine and strychnine in tonic doses may be given. Cocaine or the fluid extract of coca has been recommended as a substitute for alcohol, but it is not of much service. Prolonged seclusion in a suitable institution is in reality the only

care of Gulbenkian.

In this form of Mania a potu in which

Tobacco = Toxic Influences of use of = write section on.

effectual means of cure. When the hereditary tendency is strongly developed a lapse into the drinking habits is almost inevitable.

In delirium tremens the patient should be confined to bed and carefully watched night and day. The danger of escape in these cases is very great, as the patient imagines himself pursued by enemies or demons. Flint mentions the case of a man who escaped in his night-clothes and ran barefooted for fifteen miles on the frozen ground before he was overtaken. The patient should not be strapped in bed, as this aggravates the delirium; sometimes, however, it may be necessary, in which case a sheet tied across the bed may be sufficient, and this is certainly better than violent restraint by three or four men. Alcohol should be withdrawn at once unless the pulse is feeble.

Delirium tremens is a disease which, in a large majority of cases, runs a course very slightly influenced by medicine. The indications for treatment are to procure sleep and to support the strength. In mild cases half a drachm of bromide of potassium combined with tincture of capsicum may be given every three hours. Chloral is often of great service, and may be given without hesitation unless the heart's action is feeble. Good results sometimes follow the hypodermic use of hyoscine, one one-hundredth of a grain. Opium must be used cautiously. A special merit of Ware's work was the demonstration that on a rational or expectant plan of treatment the percentage of recovery was greater than with the indiscriminate use of sedatives, which had been in vogue for many years. When opium is indicated it should be given as morphia, hypodermically. The effect should be carefully watched, and if after three or four quarter-grain doses have been given the patient is still restless and excited, it is best not to push it farther. When fever is present the tranquillizing effects of a cold douche or cold bath may be tried, or the cold pack. The large doses of digitalis formerly employed are not advisable.

Careful feeding is the most important element in the treatment of these cases. Milk and concentrated broths should be given at stated intervals. If the pulse becomes rapid and shows signs of flagging alcohol may be given in combination with the aromatic spirits of ammonia.

II. MORPHIA HABIT (*Morphiomania; Morphinism*).

This habit arises from the constant use of morphia—taken at first, as a rule, for the purpose of allaying pain. The craving is gradually engendered, and the habit in this way acquired. The injurious effects vary very much, and in the East, where opium-smoking is as common as tobacco-smoking with us, the ill effects are, according to good observers, not so striking.

The habit is particularly prevalent among women and physicians who use the hypodermic syringe for the alleviation of pain, as in neuralgia or

sciatica. The acquisition of the habit as a pure luxury is rare in this country.

The symptoms at first are slight, and moderate doses may be taken for months without serious injury and without disturbance of health. There are exceptional instances in which for a period of years excessive doses have been taken without deterioration of the mental or bodily functions. As a rule, the dose necessary to obtain the desired sensations has gradually to be increased. As the effects wear off the victim experiences sensations of lassitude and mental depression, accompanied often with slight nausea and epigastric distress, symptoms which are relieved by another dose of the drug. The confirmed opium-eater presents a very characteristic appearance. There is a sallowness of the complexion which is almost pathognomonic, and he becomes emaciated, gray, and prematurely aged. He is restless, irritable, and unable to remain quiet for any time. Itching is a common symptom. The sleep is disturbed, the appetite and digestion are deranged, and except when directly under the influence of the drug the mental condition is one of depression. Occasionally there are profuse sweats, which may be preceded by chills. The pupils, except when under the direct influence of the drug, are dilated, sometimes unequal. Persons addicted to morphia are inveterate liars, and no reliance whatever can be placed upon their statements. In many instances this is not confined to matters relating to the vice. In women the symptoms may be associated with those of pronounced hysteria or neurasthenia. The practice may be continued for an indefinite time, usually requiring increase in the dose until ultimately enormous quantities may be needed to obtain the desired effect. Finally a condition of asthenia is induced, in which the victim takes little or no food and dies from the extreme bodily debility.

The *treatment* of the morphia habit is extremely difficult, and can rarely be successfully carried out by the general practitioner. Isolation, systematic feeding, and gradual withdrawal of the drug are the essential elements. As a rule, the patients must be under control in an institution and should be in bed for the first ten days. It is best in a majority of cases to reduce the morphia gradually. The diet should consist of beef-juices, milk, and egg-white, which should be given at short intervals. The sufferings of the patients are usually very great, more particularly the abdominal pains, sometimes nausea and vomiting, and the distressing restlessness. Usually within a week or ten days the opium may be entirely withdrawn. In all cases the pulse should be carefully watched and, if feeble, stimulants should be given, with the aromatic spirits of ammonia and digitalis. For the extreme restlessness a hot bath is serviceable. The sleeplessness is the most distressing symptom, and various drugs may have to be resorted to, particularly hyoscine and sulphonal and sometimes, if the insomnia persists, morphia itself.

It is essential in the treatment of a case to be certain that the patient

has no means of obtaining morphia. Even under the favorable circumstances of seclusion in an institution, and constant watching by a night and a day nurse, I have known a patient to practice deception for a period of three months. After an apparent cure the patients are only too apt to lapse into the habit.

The condition is one which has become so common, and is so much on the increase, that physicians should exercise the utmost caution in prescribing morphia, particularly to female patients. Under no circumstances whatever should a patient with neuralgia or sciatica be allowed to use the hypodermic syringe, and it is even safer not to intrust this dangerous instrument to the hands of the nurse.

III. LEAD-POISONING (*Plumbism; Saturnism*).

Etiology.—The disease is wide-spread, particularly in lead-workers and among plumbers, painters, and glaziers. The metal is introduced into the system in many forms. Miners usually escape, but those engaged in the smelting of lead-ores are often attacked. Animals in the neighborhood of smelting furnaces have suffered with the disease, and even the birds that feed on the berries in the neighborhood may be affected. Men engaged in the white-lead factories are particularly prone to plumbism. Accidental contamination may come in many ways; most commonly by drinking water which has passed through lead pipes or been stored in lead-lined cisterns. Wines and cider which contain acids quickly become contaminated in contact with lead. It was the frequency of colic in certain of the cider districts of Devonshire which gave the name Devonshire colic, as the frequency of it in Poitou gave the name *colica Pictonum*. Among the innumerable sources of accidental contamination may be mentioned milk, various sorts of beverages, hair dyes, false teeth, and thread. A serious outbreak of lead-poisoning, which was investigated by David D. Stewart, occurred recently in Philadelphia, owing to the disgraceful adulteration of a baking-powder with chromate of lead, which was used to give a yellow tint to the cakes. Lead given medicinally rarely produces poisoning.

All ages are attacked, but J. J. Putnam states that children are relatively less liable. The largest number of cases occur between thirty and forty. According to Oliver, from whose recent Gulstonian lectures I here quote, females are more susceptible than males. He states that they are much more quickly brought under its influence, and in a recent epidemic in which a thousand cases were involved the proportion of females to males was four to one.

The lead gains entrance to the system through the lungs, the digestive organs, or the skin. Poisoning may follow the use of cosmetics containing lead. Through the lungs it is freely absorbed. The chief channel,

according to Oliver, is the digestive system. It is rapidly eliminated by the kidneys and skin, and is present in the urine of lead-workers. The susceptibility is remarkably varied. The symptoms may be manifest with a month of exposure. On the other hand, Tanquerel (des Planches) met with a case in a man who had been a lead-worker for fifty-two years.

Morbid Anatomy.—Small quantities of lead occur in the body in health. J. J. Putnam's reports show that of 150 persons not presenting symptoms of lead-poisoning traces of lead occurred in the urine of 25 per cent.

In chronic poisoning lead is found in the various organs. The affected muscles are yellow, fatty, and fibroid. The nerves present the features of a peripheral degenerative neuritis. The cord and the nerve-roots are, as a rule, uninvolved. In the primary atrophic form the ganglion cells of the anterior horns are probably involved. In the acute fatal cases there may be the most intense entero-colitis.

Clinical Forms.—*Acute Poisoning.*—We do not refer here to the accidental or suicidal cases, which present vomiting, pain in the abdomen, and collapse symptoms. In workers in lead there are several manifestations which follow a short time after exposure and set in acutely. There may be, in the first place, a rapidly developing anæmia. Acute neuritis has been described, and convulsions, epilepsy, and a delirium, which may be, as Stephen Mackenzie has noted, not unlike that produced by alcohol. There are also cases in which the gastro-intestinal symptoms are most intense and rapidly prove fatal. There was admitted under my care in the Philadelphia Hospital a painter, aged fifty, suffering with anæmia and severe abdominal pain, which had lasted about a week. He had vomiting, constipation at first, afterward severe diarrhœa and melæna, with distention and tenderness of the abdomen. There were albumen and tube-casts in the urine. The temperature was usually subnormal. Death occurred at the end of the second week. There was found the most intense entero-colitis with hæmorrhages and exudation. These acute forms develop more frequently in persons recently exposed, and, according to Mackenzie, are more frequent in winter than in summer.

Chronic poisoning presents the following symptoms:

(*a*) *Anæmia*, the so-called saturnine cachexia, which may be profound. As a rule, however, the corpuscles do not sink below 50 per cent.

(*b*) *Blue line* on gums, which is a valuable indication, but not invariably present. Two lines must be distinguished: one, at the margin between the gums and teeth, is on, not in the gums, and is readily removed by rinsing the mouth and cleansing the teeth. The other is the well-known characteristic blue-black line at the margin of the gum. The color is not uniform, but being in the papillæ of the gums the line is, as seen with a magnifying-glass, interrupted. The lead is absorbed and converted in the tissues into a black sulphide by the action of sulphuretted hydrogen from the tartar of the teeth. The line may form rapidly after exposure and

disappear within a few weeks, or may persist for many months. Philipson has noted the occurrence of a black line in miners, due to the deposition of carbon.

The most important symptoms of chronic lead-poisoning are colic, lead-palsy, and the encephalopathy. Of these, the colic is the most frequent. Of Tanquerel's cases, there were 1,217 of colic, 101 of paralysis, and 72 of encephalopathy.

(*c*) *Colic* is the most common symptom of chronic lead-poisoning. It is often preceded by gastric or intestinal symptoms, particularly constipation. The pain is over the whole abdomen. The colic is usually paroxysmal, like true colic, and is relieved by pressure. There is often, in addition, between the paroxysms a dull, heavy pain. There may be vomiting. During the attack, as Riegel noted, the pulse is increased in tension and the heart's action is retarded. The pupils are usually unequal (Oliver).

(*d*) *Lead-palsy.*—This is rarely a primary manifestation. The onset may be acute, subacute, or chronic. It usually develops without fever. In its distribution it may be partial, limited to a muscle or to certain muscle groups, or generalized, involving in a short time the muscles of the extremities and the trunk. Madame Déjérine-Klumpke recognizes the following *localized forms:*

(1) Anti-brachial type, paralysis of the extensors of the fingers and of the wrist. In this the musculo-spiral nerve is involved, causing the characteristic wrist-drop. The supinator longus usually escapes.

(2) Brachial type, which involves the deltoid, the biceps, the brachialis anticus, and the supinator longus, rarely the pectorals. The atrophy is of the scapulo-humeral form. It is bilateral, and sometimes follows the first form, but it may be primary.

(3) The Aran-Duchenne type, in which the small muscles of the hand and of the thenar and hypothenar eminences are involved. It produces a paralysis closely resembling that of the early stage of *polio-myelitis anterior chronica.* The atrophy is marked, and may be the first manifestation of the lead-palsy. Möbius has shown that this form is particularly developed in tailors.

(4) The peroneal type. According to Tanquerel, the lower limbs are involved in the proportion of thirteen to one hundred of the upper limbs. The lateral peroneal muscles, the extensor communis of the toes, and the extensor proprius of the big toe are involved, producing the *steppage* gait.

(5) Laryngeal form. Adductor paralysis has been noted by Morell Mackenzie and others in lead-palsy.

Generalized Palsies.—There may be a slow, chronic paralysis, gradually involving the extremities, beginning with the classical picture of wrist-drop. More frequently there is a rapid generalization, producing complete paralysis in all the muscles of the parts in a few days. It may pursue a course like an ascending paralysis, associated with rapid wasting

of all four limbs. Such cases, however, are very rare. Death has occurred by involvement of the diaphragm. Oliver reports a case of Philipson's in which complete paralysis supervened. Déjérine-Klumpke also recognizes a febrile form of general paralysis in lead-poisoning, which may closely resemble the subacute spinal paralysis of Duchenne.

There is also a primary saturnine muscular atrophy in which the weakness and wasting come on together and develop proportionately. It is this form, according to Gowers, which most frequently assumes the Aran-Duchenne type.

The electrical reactions are those of lesions of the lower motor segment, and have been described under lesions of the nerves. The degenerative reaction in its different grades may be present, depending upon the severity of the disease.

Usually with the onset of the paralysis there are pains in the legs and joints, the so-called saturnine arthralgias. As a rule, however, sensation is unaffected and the sensory nerves are not involved.

(*e*) The *cerebral symptoms* are numerous. Optic neuritis or neuroretinitis may develop. Hysterical symptoms occasionally occur in girls. Epilepsy is not uncommon, and in fits developing in the adult the possibility of lead-poisoning should always be considered. An acute delirium may occur with hallucinations. The patients may have trance-like attacks, which follow or alternate with convulsions. A few cases of lead encephalopathy finally drift into lunatic asylums. Tremor is one of the commonest manifestations of lead-poisoning.

(*f*) *Arterio-sclerosis.*—Lead-workers are notoriously subject to arterio-sclerosis with contracted kidneys and hypertrophy of the heart. The cases usually show distinct gouty deposits, particularly in the big-toe joint; but in this country acute gout in lead-workers is rare. According to Sir William Roberts, the lead favors the precipitation of the crystalline urates of the tissues. Ralfe has shown that lead diminishes the alkalinity of the blood, and so lessens the solubility of the uric acid.

Prognosis.—In the minor manifestations of lead-poisoning this is good. According to Gowers, the outlook is bad in the primary atrophic form of paralysis. Convulsions are, as a rule, serious, and the mental symptoms which succeed may be permanent. Occasionally the wrist-drop persists.

Treatment.—Prophylactic measures should be taken at all lead-works, but unless employés are careful poisoning is apt to occur even under the most favorable conditions. Cleanliness of the hands and of the finger-nails, frequent bathing, and the use of respirators when necessary, should be insisted upon. When the lead is in the system, the iodide of potassium should be given in from five- to ten-grain doses three times a day. For the colic, local applications and, if severe, morphia may be used. An occasional morning purge of sulphate of magnesia may be given. For the anæmia iron should be used. In the very acute cases it is well not

however
may be
convulsions are

True epilepsy may follow the convulsions.

(See D. Stewart. Am. J. March '55.

The arsenic compounds may be either in the form of solid particles detached from the paper or as gaseous volatile body
The investigations of Gosio confirmed by Sanger, have shown that
a volatile compound by the action of arsenical organic
matter in wall papers of several moulds, notably Penicillium
brevicaule, Mucor mucedo etc In moisture & at a temperature
from 60 - 95°F these bodies set free a volatile compound
probably "an organic derivative of arsenic pentoxide" (Sanger)
The chronic poisoning from fabrics & wall papers may be
due according to this author to the ingestion of minute amounts
of this derivative which from its stable form oxidation is likely
to be accumulated in the system from which it is slowly eliminated"

In chorea neurotic has occurred after.
A Dr patient of mine with Hodgkins Disease developed multiple
. arsenical neuritis after taking 3 i ℥ of Asthmolers
solution in minutes

to give the iodide, as, according to some writers, the liberation of the lead which has been deposited in the tissues may increase the severity of the symptoms. For the local palsies massage and the constant current should be used.

IV. ARSENICAL POISONING.

Acute poisoning by arsenic is common, particularly by Paris green and such mixtures as "Rough on Rats," which are used to destroy vermin and insects. The chief symptoms are intense pain in the stomach, vomiting, and, later, colic, with diarrhœa and tenesmus; occasionally the symptoms are those of collapse. If recovery takes place, paralysis may follow. The treatment should be similar to that of other irritant poisons—rapid removal with the stomach pump, the promotion of vomiting, and the use of milk and eggs. If the poison has been taken in solution, dialyzed iron may be used in large doses of from six to eight drachms.

Chronic Arsenical Poisoning.—Arsenic is used extensively in the arts, particularly in the manufacture of colored papers, artificial flowers, and in many of the fabrics employed as clothing. The glazed green and red papers used in kindergartens also contain arsenic. It is present also in many wall-papers and carpets. Much attention has been paid to this question of late years, as instances of poisoning have been thought to depend upon wall-papers and other household fabrics. ~~According to J. J. Putnam, the greatest danger is from the dust blown off by currents of air or detached by the brush. It is thought, too, that possibly some volatile compound of arsenic may be formed~~. Arsenic is eliminated in all the secretions, and has been found in the milk. J. J. Putnam, it should be remembered, has shown that it is not uncommon to find traces of arsenic in the urine of many persons in apparent health. The effects of moderate quantities of arsenic are not infrequently seen in medical practice. In chorea and in pernicious anæmia, steadily increasing doses are often given until the patient takes from fifteen to twenty drops of Fowler's solution three times a day. Flushing and hyperæmia of the skin, puffiness of the eyelids or above the eyebrows, nausea, vomiting, and diarrhœa are the most common symptoms. Redness and sometimes bleeding of the gums and salivation occur. In the protracted administration of arsenic patients may complain of numbness and tingling of the fingers. ~~In the large number of patients to whom I have administered arsenic, often in doses which might be termed excessive, I have seen only one case in which numbness and tingling were marked.~~ Pigmentation of the skin I have seen on several occasions.

In the slow poisoning by the absorption of arsenic in minute doses, as from wall-paper and fabrics, the symptoms are varied. J. J. Putnam groups them into the cases in which the symptoms mainly concern the general nutrition without signs of local irritation; those in which the symptoms

are due to irritation of the conjunctivæ, mouth, or pharynx; those with symptoms pointing to the digestive tract; cases with marked nervous phenomena; and those in which the nutrition of some special part of the body is involved. The most common symptoms are those of anæmia and debility, perhaps with slight irritation of the mucous membrane, and numbness and tingling. How far these symptoms are to be attributed to the small quantities of arsenic absorbed from wall-papers and fabrics is by some considered doubtful. That children and adults may take with impunity large doses for months without unpleasant effects, and the fact of the gradual establishment of a toleration which enables Styrian peasants to take as much as eight grains of arsenious acid in a day, speak strongly against it.

Arsenical paralysis has the same characteristics as lead-palsy, but the legs are more affected than the arms, particularly the extensors and peroneal group, so that the patient has the characteristic *steppage* gait of peripheral neuritis.

The electrical reaction in the muscles may be disturbed before any loss of power, and when the patient is asked to extend the wrist fully and to spread the fingers slight weakness may be detected early.

V. PTOMAINE POISONING.

In the bacterial decomposition of animal matters chemical compounds are formed, the putrefactive alkaloids, known as ptomaines and toxines, some of which are highly poisonous. They differ extraordinarily in their chemical characters and physiological effects. Some only are poisonous, and these Brieger has designated as toxines. The specific action of the micro-organisms in disease is now attributed in large part to the formation of these bodies, and the whole question of immunity and protection is now being worked out in this direction, a special stimulus having been given of late in the discovery by Hankin of the so-called defensive alkaloids (see under pneumonia).

Our interest here is in the effects of these poisons when taken with foods.*

It is quite possible that the leucomaines, the basic substances formed in the living body, may under certain circumstances be capable of causing disease. Products also of the bacterial decomposition in the intestines may be absorbed and act as poisons. Our knowledge on these points is as yet scanty and uncertain. A suggestive chapter (XIII) upon the subject is to be found in the work of Vaughan and Novy.

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* For a full discussion of the whole subject the student is referred to the Manual upon Ptomaines and Leucomaines, by Vaughan and Novy, second edition, Philadelphia, 1891.

and gastralgia

On the other hand as Sanger states we do not know accurately the effects of many of the compounds in minute & long continued doses, notably of the arsenicals.

Brass-workers diseases &c

Among the more common forms are the following:

(1) **Meat Poisoning.**—Cases have usually followed the eating of sausages or pork-pie or head-cheese, and also occasionally beef, veal, and mutton. Sausage poisoning, which is known by the name of *botulism* or *allantiasis*, has long been recognized, and there have been numerous outbreaks, particularly in parts of Germany. Similar attacks have been produced by ham and by head-cheese. The precise nature of the poison in these cases has not yet been determined. Other outbreaks have followed the eating of beef and veal. In the majority of these cases the meat has undergone decomposition, though the change may not have been evident to the taste. The symptoms of meat poisoning are those of acute gastro-intestinal irritation. Ballard's description of the Wellbeck cases, quoted by Vaughan, holds good for a majority of them:

"A period of incubation preceded the illness. In 51 cases where this could be accurately determined, it was twelve hours or less in 5 cases; between twelve and thirty-six hours in 34 cases; between thirty-six and forty-eight hours in 8 cases; and later than this in only 4 cases. In many cases the first definite symptoms occurred suddenly, and evidently unexpectedly, but in some cases there were observed during the incubation more or less feeling of languor and ill-health, loss of appetite, nausea, or fugitive, griping pains in the belly. In about a third of the cases the first definite symptom was a sense of chilliness, usually with rigors, or trembling, in one case accompanied by dyspnœa; in a few cases it was giddiness with faintness, sometimes accompanied by a cold sweat and tottering; in others the first symptom was headache or pain somewhere in the trunk of the body—e. g., in the chest, back, between the shoulders, or in the abdomen, to which part the pain, wherever it might have commenced, subsequently extended. In one case the first symptom noticed was a difficulty in swallowing. In two cases it was intense thirst. But however the attack may have commenced, it was usually not long before pain in the abdomen, diarrhœa, and vomiting came on, diarrhœa being of more certain occurrence than vomiting. The pain in several cases commenced in the chest or between the shoulders, and extended first to the upper and then to the lower part of the abdomen. It was usually very severe indeed, quickly producing prostration or faintness, with cold sweats. It was variously described as crampy, burning, tearing, etc. The diarrhœal discharges were in some cases quite unrestrainable, and (where a description of them could be obtained) were said to have been exceedingly offensive and usually of a dark color. Muscular weakness was an early and very remarkable symptom in nearly all the cases, and in many it was so great that the patient could only stand by holding on to something. Headache, sometimes severe, was a common and early symptom; and in most cases there was thirst, often intense and most distressing. The tongue, when observed, was described usually as thickly coated with a brown, velvety fur, but red at the tip and edges. In the early stage the skin was often cold to the

touch, but afterward fever set in, the temperature rising in some cases to 101°, 103°, and 104° F. In a few severe cases, where the skin was actually cold, the patient complained of heat, insisted on throwing off the bed-clothes, and was very restless. The pulse in the height of the illness became quick, counting in some cases 100 to 128. The above were the symptoms most frequently noted. Other symptoms occurred, however, some in a few cases, and some only in solitary cases. These I now proceed to enumerate. Excessive sweating, cramps in the legs, or in both legs and arms, convulsive flexion of the hands or fingers, muscular twitchings of the face, shoulders, or hands, aching pain in the shoulders, joints, or extremities, a sense of stiffness of the joints, prickling or tingling or numbness of the hands lasting far into convalescence in some cases, a sense of general compression of the skin, drowsiness, hallucinations, imperfection of vision, and intolerance of light. In three cases (one that of a medical man) there was observed yellowness of the skin, either general or confined to the face and eyes. In one case, at a late stage of the illness, there was some pulmonary congestion and an attack of what was regarded as gout. In the fatal cases death was preceded by collapse like that of cholera, coldness of the surface, pinched features, and blueness of the fingers and toes and around the sunken eyes. The debility of convalescence was in nearly all cases protracted to several weeks.

"The mildest cases were characterized usually by little remarkable beyond the following symptoms, viz., abdominal pains, vomiting, diarrhœa, thirst, headache, and muscular weakness, any one or two of which might be absent."

Many instances are on record of poisoning by canned goods, particularly meat. Some of these, according to John G. Johnson, have been cases of corrosive poisoning from muriate of zinc and muriate of tin used as an amalgam, but poisonous effects identical with those just described have followed the use of canned meats.

Certain game birds, particularly the grouse, are stated to be poisonous, in special districts and at certain seasons of the year.

(2) **Poisoning by Milk Products.**—Poisoning by cheese has long been known. In Michigan, in 1883 and 1884, there were nearly 300 cases of cheese poisoning, and from pieces of the cheese Vaughan separated a substance which he called tyrotoxicon. Since that date other outbreaks have been reported. Apparently to this poison also are due the outbreaks following the use of milk, several of which are reported in the manual by Vaughan and Novy. Still more numerous of late years have been the cases due to poisonous ice-cream, in which also the tyrotoxicon has been found.

The symptoms are those of acute gastro-intestinal irritation, and are similar to those already detailed by Ballard.

(3) **Poisoning by Shell-fish and Fish.**—Perhaps the most serious form of *ichthysmus*, as the disease is called, is that produced by the mussel,

many epidemics of which have been studied of late, more particularly an outbreak at Wilhelmshaven. Brieger has separated a poison which he has called *mytilotoxin*. It has been shown that this exists chiefly in the liver of the mussel. It does not yet appear to be settled whether there is a special poisonous variety or whether the mussel only becomes toxic under certain conditions. The latter seems to be the most probable view, as Schmiltmann found that the non-poisonous mussels soon became toxic when placed in the Wilhelmshaven bay, while those from the bay soon lost their toxic properties when placed in the open sea.

The symptoms of mussel poisoning follow the eating of either raw or cooked mussels. The symptoms are those of an acute poisoning with profound action on the nervous system, and without gastro-intestinal symptoms. There are numbness and coldness, no fever, dilated pupils, rapid pulse, and death occurs sometimes within two hours with collapse symptoms.

Poisoning occasionally follows the eating of oysters which are stale or decomposed. The symptoms are usually gastro-intestinal. Certain fish also cause poisoning, more particularly the salted sturgeon used in parts of Russia, which has sometimes proved fatal to large numbers of persons. In the middle parts of Europe the barb is stated to be sometimes poisonous, producing the so-called "*barbus cholera*." In China and Japan various species of the *tetrodon* are also toxic, sometimes proving fatal within an hour, with symptoms of intense disturbance of the nervous system. Several other poisonous forms are known, which produce symptoms described as *ichthyismus paralyticus*.

VI. GRAIN POISONING.

(1) **Ergotism.**—The prolonged use of meal made from grains contaminated with the ergot fungus (*clavicaps purpurea*) causes a series of symptoms known as ergotism, epidemics of which have prevailed in different parts of Europe. Two forms of this chronic ergotism are described—the gangrenous and the convulsive or spasmodic. In the former, mortification affects the extremities—usually the toes and fingers, less commonly the ears and nose. Preceding the onset of the gangrene there are usually anæsthesia, tingling, pains, spasmodic movements of the muscles, and gradual blood stasis in certain vascular territories.

The nervous manifestations are very remarkable. After a prodromal stage of ten to fourteen days, in which the patient complains of weakness, headache, and tingling sensations in different parts of the body, perhaps accompanied with slight fever, spasmodic symptoms develop, producing cramps in the muscles and contractures. The arms are flexed and the legs and toes extended. These spasms may last from a few hours to many days and relapses are frequent. In severe cases epilepsy develops and the

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patient may die in convulsions. Mental symptoms are common, manifested sometimes in a preliminary delirium, but more commonly, in the chronic poisoning, as melancholia or dementia. Posterior spinal sclerosis occurs in chronic ergotism. In the interesting group of 29 cases studied by Tuczek and Siemens, nine died at various periods after the infection, and four post-mortems showed degeneration of the posterior columns. A condition similar to tabes dorsalis is gradually produced by this slow degeneration in the spinal cord.

(2) **Lathyrism** (*Lupinosis*).—An affection produced by the use of meal from varieties of vetches, chiefly the *Lathyrus sativus* and *L. cicera.* The grain is popularly known as the chick-pea. The grains are usually powdered and mixed with the meal from other cereals in the preparation of bread. As early as the seventeenth century it was noticed that the use of flour with which the seeds of the *Lathyrus* were mixed caused stiffness of the legs. The subject did not, however, attract much attention until the studies of James Irving, in India, who between 1859 and 1868 published several important communications, describing a form of spastic paraplegia affecting large numbers of the inhabitants in certain regions of India and due to the use of meal made from the *Lathyrus* seeds. It also produces a spastic paraplegia in animals. The Italian observers describe a similar form of paraplegia, and it has been observed in Algiers by the French physicians. The condition is that of a spastic paralysis, involving chiefly the legs, which may proceed to complete paraplegia. The arms are rarely, if ever, affected. It is evidently a slow sclerosis induced under the influence of this toxic agent. The precise anatomical condition, so far as I can ascertain, has not yet been determined.

(3) **Pellagra.**—This is a nutritional disturbance due to the use of altered maize. The disease occurs extensively in parts of Italy, in the south of France, and in Spain. It has not been observed in this country. It prevails extensively among the poorer classes, particularly in the country districts, and appears to be associated in some way with the use of maize which (according to most authorities) is fermented or diseased. In the early stage the symptoms are indefinite, characterized by debility, pains in the spine, insomnia, digestive disturbances, more rarely diarrhœa. The first clear manifestation of the disease is the pellagral erythema, which almost invariably appears in the spring. This is followed by desiccation and exfoliation of the epidermis, which becomes very rough and dry, and occasionally crusts form, beneath which there is suppuration. With these cutaneous manifestations there are digestive troubles—salivation, dyspepsia, and diarrhœa—which may be of a dysenteric nature. After lasting for a few months improvement occurs in the milder cases and convalescence is gradually established. In the more severe and chronic forms there are pronounced nervous symptoms—headache, backache, spasms, and finally paralysis and mental disturbance. The paralytic condition affects the legs and leads gradually to paraplegia. The mental manifestations, which

are rarely met with until the third or fourth attack, are melancholia or suicidal mania. Finally, there may be a condition of the most pronounced cachexia.

The anatomical changes are indefinite. Chronic degenerative changes have been found, particularly fatty degeneration and a peculiar pigmentation in the viscera. The measures to be employed are change in diet, removal from the infected district, and, as a prophylaxis, proper preservation of the maize.*

VII. SUNSTROKE

(*Heat Exhaustion; Insolation; Thermic Fever; Heat-stroke; Coup de Soleil*).

Definition.—A condition produced by exposure to excessive heat.

It is one of the oldest of recognized diseases; two instances are mentioned in the Bible. It was long confounded with apoplexy. The Anglo-Indian surgeons gave admirable descriptions of it. In this country the most important contributions have come from the New York Hospital and the Pennsylvania Hospital; from the former, the studies of Swift and Darrach, from the latter, the papers of Gerhard, George B. Wood, the elder Pepper, and Levick. In New Orleans, Bennett Dowler studied the disease and recognized the difference between heat exhaustion and sunstroke. Very little has been added to our knowledge of the disease since the publication of a monograph by H. C. Wood. Two forms are recognized, heat exhaustion and heat-stroke.

Heat Exhaustion.—Prolonged exposure to high temperatures, particularly when combined with physical exertion, is liable to be followed by extreme prostration, collapse, restlessness, and in severe cases by delirium. The surface is usually cool, the pulse small and rapid, and the temperature may be subnormal—as low as 95° or 96°. The individual need not necessarily be exposed to the direct rays of the sun, but the condition may come on when working in close, confined rooms during midsummer. It may also follow exposure to great artificial heat; thus the stokers in the Atlantic steamships sometimes succumb to the effect of the great heat in the engine rooms.

Sunstroke or Thermic Fever.—The cases are chiefly found in persons who, while working very hard, are exposed to the sun. Soldiers on the march with their heavy accoutrements are particularly liable to attack. In the larger cities of this country the cases are almost exclusively confined to workmen who are much exposed and, at the same time, have been drinking beer and whisky.

Morbid Anatomy and Pathology.—*Rigor mortis* occurs early. Putrefactive changes develop with great rapidity. The venous engorge-

* The most elaborate discussion of the subject is by Jules Arnould in the Dictionnaire Encyclopédique des Sciences Médicales, tome xxii, 1886.

ment is extreme, particularly in the cerebrum. The left ventricle is contracted (Wood), and the right chamber dilated. The blood is usually fluid; the lungs are intensely congested. Parenchymatous changes occur in the liver and kidneys.

According to Wood, "heat exhaustion with lowered temperature represents a sudden vaso-motor palsy, i. e., a condition in which the existing effect of the heat paralyzes the centre in the medulla." On the other hand, thermic fever is held to be due to paralysis under the influence of the extreme external heat of the centre in the medulla which regulates the disposition of the bodily heat. Owing to this disturbance, more heat is produced and less given off than normally.

Symptoms.—The patient may be struck down and die within an hour with symptoms of heart failure, dyspnœa, and coma. This form, sometimes known as the asphyxial, occurs chiefly in soldiers and is graphically described by Parkes. Death indeed may be almost instantaneous, the victims falling as if struck upon the head. The usual form in this latitude comes on during exposure, with pain in the head, dizziness, a feeling of oppression, and sometimes nausea and vomiting. Visual disturbances are common, and a patient may have colored vision. Diarrhœa or frequent micturition may supervene. Insensibility follows, which may be transient or which deepens into a profound coma. The patients are usually admitted to hospital in an unconscious state, with the face flushed, the skin pungent, the pulse rapid and full, and the temperature ranging from 107° to 110°, or even higher. F. A. Packard states that of the 31 cases admitted to the Pennsylvania Hospital in the summer of 1887, in a majority of them the temperature was between 110° and 111°. In one case the temperature was 112°. The breathing is labored and deep, sometimes stertorous. Usually there is complete relaxation of the muscles, but twitchings, jactitation, or very rarely convulsions may occur. The pupils may at first be dilated, but by the time the cases are admitted to hospital they are (in a majority) extremely contracted. Petechiæ may be present upon the skin. In the fatal cases the coma deepens, the cardiac pulsations become more rapid and feeble, the breathing becomes hurried and shallow and of the Cheyne-Stokes type. The fatal termination may occur within twenty-four or thirty-six hours. Favorable indications are the recovery of consciousness and a fall in the fever. The recovery in these cases may be complete. In other instances there are remarkable after-effects, the most constant of which is a permanent inability to bear high temperatures. Such patients become very uneasy when the thermometer reaches 80° F. in the shade. An extraordinary instance came under my notice in which the patient was subsequently so sensitive to temperatures in the neighborhood of 75° F. that at such times he lived comfortably only in the cellar, and finally sought refuge in Alaska. Loss of the power of mental concentration and failure of memory are more constant and very troublesome sequelæ. Such patients are always worse

For the convulsions chloroform should be given at once.

"An only daughter" in the words of Lord Byron

in the hot weather. Occasionally convulsions and marked mental disturbance may develop. H. C. Wood states that in a case of this kind chronic meningitis was found.

Guitéras has called attention to a form of fever occurring in the South, known in Florida as "Florida fever," in the Carolinas as "country fever," and in tropical countries as *fièvre inflammatoire* The cases last for a variable time, and are mistaken for malaria or typhoid; but he believes them to be entirely distinct and due to a prolonged action of the high temperatures. He has called the condition a "continued thermic fever."

The diagnosis of heat exhaustion from thermic fever is readily made, as the difference between the two conditions is striking. "In solar exhaustion the skin is moist, pale, and cool; the breathing is easy though hurried; the pulse is small and soft; the vital forces fall into a temporary collapse; the senses remain entire" (Dowler); whereas in sunstroke or heat apoplexy there is usually unconsciousness and pyrexia.

The mode of onset, together with the circumstances under which it occurs and the high temperature, permits thermic fever to be readily differentiated from apoplexy, and coma from other conditions.

Treatment.—In heat exhaustion stimulants should be given freely, and if the temperature is below normal the hot bath should be used. Ammonia may be given if necessary. In thermic fever the indications are to reduce the temperature as rapidly as possible. This may be done by placing the patient in a bath at 70°. Rubbing the body with ice was practised at the New York Hospital by Darrach in 1857, and is an excellent procedure to lower the temperature rapidly. Ice-water enemata may also be employed. At the Pennsylvania Hospital in the summer of 1887 the ice-pack was used with great advantage. Of 31 cases only 12 died, a result probably as satisfactory as can be obtained, considering that many of the patients are almost moribund when brought to hospital. It should be compared with Swift's statistics, in which of 150 cases 78 died. In the cases in which the symptoms are those of intense asphyxia, and in which death may take place in a few minutes, free bleeding should be practised, a procedure which saved Weir Mitchell when a young man. Of other remedies, the antipyretics have been employed, and may be given when there is any special objection to hydrotherapy, for which, however, they cannot be substituted.

VIII. OBESITY.

Corpulence, an excessive development of the bodily fat, is a condition for which the physician is frequently consulted, and for which much may be done by a judicious arrangement of the diet. The tendency to polysarcia or obesity is often hereditary, and is particularly apt to be manifest after the middle period of life. It may, however, be seen early, and in this country it is not very uncommon in young girls and young boys.

A very important factor is overeating, a vice which is more prevalent and only a little behind overdrinking in its disastrous effects. A majority of persons over forty years of age habitually eat too much. In some of the most aggravated cases of obesity, however, this plays no part, and the unfortunate victim may be a notoriously small eater. A second element is lack of proper exercise; a third less important factor is the taking largely of alcoholic beverages, particularly beer.

In obesity it is now generally conceded that the carbohydrates, which were so long blamed, are not at fault, since they are themselves converted into water and carbon dioxide. On account, however, of the facility with which they are utilized for the purposes of oxidation the albuminous elements of the food are less readily oxidized, not so fully decomposed, and the fat is in reality separated from them. So, too, the fats themselves are not so prone to cause obesity as the carbohydrates, being less readily oxidized and interfering less with the complete metabolism of the albuminous elements.

Many plans are now advised for the reduction of fat, the most important of which are those of Banting, Ebstein, and Oertel. In the Banting method the amount of food is reduced, the liquids are restricted, and the fats and carbohydrates excluded.

Ebstein recommends the use of fat and the rapid exclusion of the carbohydrates. The following is an example of his dietary:

Breakfast (6 A. M. in summer, 7.30 A. M. in winter).—White bread, well toasted (rather less than two ounces) and well covered with butter. Tea, without milk or sugar, eight or nine ounces.

Dinner, 2 P. M.—Soup made with beef-marrow. Fat meat, with fat sauce, four to five ounces. A moderate quantity of asparagus, spinach, cabbage, peas, and beans. Two or three glasses of light white wine. After the meal, a large cup of tea without milk or sugar.

Supper, at 7.30 P. M.—An egg, a little roast meat, with fat. About an ounce of bread, well covered with butter. A large cup of tea, without milk or sugar.

Oertel's method has already been considered in connection with the treatment of fatty heart, and is combined with systematic bodily exercise. It is particularly adapted for stout persons with weak heart.

The so-called Schweninger cure is in reality Oertel's, with the sole modification of the forbidding of any fluid at meals. Liquids must be taken more than two hours after the food.

Yeo, after a full consideration of the various methods, gives the following useful summary:

"The albuminates in the form of animal food should be strictly limited. Farinaceous and all starchy foods should be reduced to a minimum. Sugar should be entirely prohibited. A moderate amount of fats, for the reasons given by Ebstein, should be allowed.

"Only a small quantity of fluid should be permitted at meals, but

And lastly sensible people may eat with advantage
Spaghetti
Thyme and basil in Abundance.

enough should be allowed to aid in the solution and digestion of the food. Hot water or warm aromatic beverages may be taken freely between meals or at the end of the digestive process, especially in gouty cases, on account of their eliminative action.

"No beer, porter, or sweet wines of any kind to be taken; no spirit, except in very small quantity. It should be generally recognized that the use of alcohol is one of the most common provocatives of obesity. A little Hock, still Moselle, or light claret, with some alkaline table water is all that should be allowed. The beneficial effects of such diet will be aided by abundant exercise on foot and by the free use of saline purgatives, so that we may insure a complete daily unloading of the intestinal canal.

"It is only necessary to mention a few other details. Of animal foods, all kinds of lean meat may be taken, poultry, game, fish (eels, salmon, and mackerel are best avoided), eggs.

"Meat should not be taken more than once a day, and not more than six ounces of cooked meat at a time. Two lightly boiled or poached eggs may be taken at one other meal, or a little grilled fish.

"Bread should be toasted in thin slices and completely, not browned on the surface merely.

"Hard captain's biscuits may also be taken.

"Soups should be avoided, except a few tablespoonfuls of clear soup.

"Milk should be avoided, unless skimmed and taken as the chief article of diet. All milk and farinaceous puddings and pastry of all kinds are forbidden. Fresh vegetables and fruit are permitted.

"It is important to bear in mind that the actual quantity of food permitted must have a due relation to the physical development of the individual, and that what would be adequate in one case might be altogether inadequate in the case of another person of larger physique." *

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* A System of Therapeutics, vol. i, edited by H. A. Hare, Philadelphia, 1891.

SECTION XI.

DISEASES DUE TO ANIMAL PARASITES.

I. PSOROSPERMIASIS.

UNDER this term are embraced several affections produced by the sporozoa. These parasites belonging to the lowest division of the protozoa, are also known as psorosperms and gregarinidæ. They are extraordinarily abundant in the invertebrates, and are not uncommon in the higher mammals. The entire group of blood parasites, hæmatozoa, which live within the corpuscles, are closely related to them. Psorosperms are, as a rule, parasites of the cells—*cytozoa*. The commonest and most suitable variety for study is the *coccidium oviforme* of the rabbit, which produces a disease of the liver in which the organ is studded throughout with whitish nodules, ranging in size from a pin's head to a split pea. On section each nodule is seen to be a dilated portion of a bile duct; the walls are lined with epithelium in the interior of which are multitudes of ovoid bodies—the coccidia. Another very common form occurs in the muscles of the pig, the so-called Rainey's tube, which is an ovoid body within the sarcolemma containing a number of small, sickle-shaped, unicellular organisms.

These bodies probably play a more important *rôle* in human pathology than has hitherto been thought. The cases reported may be grouped under the following divisions: internal and external.

(1) **Internal Psorospermiasis.**—In a majority of the cases of this group the psorosperms have been found in the liver, producing a disease similar to that which occurs in rabbits. In Guebler's case there were tumors which could be felt in the liver during life, and they were determined by Leuckart to be due to coccidia. In W. B. Haddon's case the patient was admitted to St. Thomas's Hospital with slight fever, drowsiness, and gradual unconsciousness; death occurred on the fourteenth day of observation. Whitish neoplasms were found upon the peritonæum, omentum, and on the layers of the pericardium; and a few were found in the liver, spleen, and kidneys. A somewhat similar case, though more remarkable, as it ran a very acute course, is reported by Silcott. A woman, aged fifty-three, admitted to St. Mary's Hospital, was thought to be suffering from typhoid

fever. She had had a chill six weeks before admission. There was fever of an intermittent type, slight diarrhœa, nausea, tenderness over the liver and spleen, and a dry tongue; death occurred from heart-failure. The liver was enlarged, weighed eighty-three ounces, and in its substance there were caseous foci, around each of which was a ring of congestion. The spleen weighed sixteen ounces and contained similar bodies. The ileum presented six papule-like elevations. The masses resembled tubercles, but on examination coccidia were found.

The parasites are also found in the kidneys and ureters. Cases of this kind have been recorded by Bland Sutton and Paul Eve. In the case reported by Eve the symptoms were hæmaturia and frequent micturition, and death took place on the seventeenth day. The nodules throughout the pelvis and ureters have been regarded as mucous cysts. In a case reported by Joseph Griffiths the tumors in the ureter caused hydronephrosis.

(2) Cutaneous Psorospermiasis.—(*a*) *Follicular.*—This remarkable skin-disease was originally described by J. C. White, under the name of *keratosis follicularis*. Darier, of Paris, has shown that this is really a parasitic affection. The lesions are chiefly on the face, the flanks, and the inguinal regions. It is at first papular, surmounted by a grayish crust, dry and hard. The lesions finally become confluent, and form a series of irregular elevations giving a rasp-like feeling to the touch.

Microscopical examination shows that in these papillomatous growths there are numerous organisms corresponding to psorosperms. At the St. Louis Hospital, in Paris, Darier was kind enough to show me the cases and specimens from whom. No one accustomed to the appearance of psorosperms as seen in the lower animals could question the truly parasitic nature of these bodies. A case of the disease has been reported in this country by [illegible]

(*b*) *Paget's Disease of the Nipple.*—In this affection, formerly regarded as an eczema, psorosperms are constantly present, as shown by Darier, A. B. Macallum, and others. They are readily demonstrated, without any special preparation, and here, too, of the nature of the bodies there can be no question.

In molluscum contagiosum and in epithelioma many observers have noted the presence of bodies which lie in and between the epithelial cells and have some resemblance to psorosperms. The bodies are readily seen in sections of epitheliomas, but they lack the sharply defined characters of the coccidia which are present in Paget's disease and in White's keratosis.

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II. DISTOMIASIS.

Several forms of trematodes or flukes are parasitic in man, and when in numbers may cause serious disease.

(1) *Liver Flukes.*—The following varieties of flukes have been found: The *distoma hepaticum*, a very common parasite in ruminants, which has a length of from twenty-eight to thirty-two millimetres. The *distoma lanceolatum*, a much smaller form, from eight to ten millimetres in length, which is also very common in sheep and cattle. The *distoma crassum*, the largest form, measuring from four to eight centimetres in length. One or two other less important forms have occasionally been met with. The studies of the Japanese physicians have brought to light the interesting fact that there is a distoma widely endemic in certain provinces in that country. Two forms have been described, the *distoma endemicum* and the *distoma perniciosum*, about which there is still a doubt whether they are different species or not. The studies of Ijima indicate that they are probably the same. According to Baelz, fully twenty per cent of the inhabitants of certain provinces are affected.

The flukes occupy the bile-passages and the upper portion of the small intestine. When in large numbers they may cause serious and fatal disease of the liver, usually with ascites and jaundice. The liver may be enormously enlarged; in Kichner's case it weighed eleven pounds. The flukes may cause a chronic cholangitis, leading to great thickening or even calcification of the walls of the bile-duct.

The endemic fluke disease of Japan is characterized by enlargement of the liver, emaciation, diarrhœa, and frequently ascites.

(2) *The Blood Fluke; Bilharzia Hæmatobia.*—This trematode is found in Egypt, southern Africa, and Arabia, and is the cause in these countries of the endemic hæmaturia. The female is about two centimetres in length, cylindrical, filiform, and about ·07 millimetre in diameter. The parasite lives in the venous system, particularly in the portal vein, and in the veins of the spleen, bladder, kidneys, and mesentery. According to Bilharz, at least fifty per cent of the lower classes in Egypt are infected with it. It is not yet known how the parasite gains entrance to the body. In all probability it is by drinking impure water containing the embryos.

The symptoms are due to changes in the mucous membrane of the urinary organs caused by the presence of the parasites in the blood-vessels of these parts. Hæmaturia is the first and most constant symptom, leading gradually to anæmia. There is generally pain during micturition. The blood is not constant in the urine. The ova of the Bilharzia are readily seen under a microscope with a low power. They are ovoid in shape, translucent, with a small spike at one end. The embryo can be readily seen.

The disease is rarely fatal; a great majority of the cases recover. Chil-

dren are more commonly attacked than grown persons, and the disease often disappears by the time of puberty.

(3) *Bronchial Fluke; Distoma Westermanii; Parasitic Hæmoptysis.*—In parts of China, Japan, and Formosa there is an epidemic disease, described by Ringer and Manson, characterized by attacks of cough and hæmoptysis associated with the presence of a small fluke in the bronchial tubes.

III. DISEASES CAUSED BY NEMATODES.

1. Ascariasis.

(*a*) *Ascaris lumbricoides*, the most common human parasite, is found chiefly in children. The female is from seven to twelve inches in length, the male from four to eight inches. The worm is cylindrical, pointed at both ends, and has a yellowish-brown, sometimes a slightly reddish color. Four longitudinal bands can be seen, and it is striated transversely. The ova, which are sometimes found in large numbers in the feces, are small, brownish-red in color, elliptical, and have a very thick covering. They measure .075 millimetre in length and .05 millimetre in width. They develop outside the body, but the life-history is not known. The parasite occupies the upper portion of the small intestine. Usually not more than one or two are present, but occasionally they exist in enormous numbers. The migrations are peculiar. They may pass into the stomach, from which they may be ejected by vomiting, or they may crawl up the œsophagus and enter the pharynx, from which they may be withdrawn. A child, under my care in the small-pox department of the General Hospital, during convalescence, withdrew in this way more than thirty round worms within a few weeks. In other instances the worm passes into the larynx, and has been known to cause fatal asphyxia, or passing into the trachea, to cause gangrene of the lung. They may pass into the Eustachian tube and appear at the external meatus. The most serious migration is into the bile-duct. There is a specimen in the Wistar-Horner Museum of the University of Pennsylvania in which not only the common duct, but also the main branches throughout the liver are enormously distended and packed with numerous round worms. [illegible] The symptoms are not definite. When a few are present they may be passed without causing disturbance. In children there are irritative symptoms usually attributed to worms, such as restlessness, irritability, picking at the nose, grinding of the teeth, twitchings, or convulsions. These symptoms may be marked in very nervous children.

Treatment.—Santonin can be given, mixed with sugar, in doses of from one to three grains for a child, and three to five grains for an adult, followed by a calomel or a saline purge. The dose may be given for

particularly boys

dung.

weslermanni

or according to some writers in the lungs near the pleura. This parasite has been found in this country by me in the cat & in dog by H B Ward.

A. Epstein Jahrb. f. Kinderheilk Leipz 1892 has proved the direct infection with the ova after keeping them for a year. The female reaches maturity on the 10th or 12th day

dren are more commonly attacked than grown persons, and the disease often disappears by the time of puberty.

(3) *Bronchial Fluke; Distoma Ringeri; Parasitic Hæmoptysis.*—In parts of China, Japan, and Formosa there is an epidemic disease, described by Ringer and Manson, characterized by attacks of cough and hæmoptysis associated with the presence of a small fluke in the bronchial tubes.

III. DISEASES CAUSED BY NEMATODES.

I. Ascariasis.

(*a*) *Ascaris lumbricoides*, the most common human parasite, is found chiefly in children. The female is from seven to twelve inches in length, the male from four to eight inches. The worm is cylindrical, pointed at both ends, and has a yellowish-brown, sometimes a slightly reddish color. Four longitudinal bands can be seen, and it is striated transversely. The ova, which are sometimes found in large numbers in the fæces, are small, brownish-red in color, elliptical, and have a very thick covering. They measure ·075 millimetre in length and ·058 millimetre in width. They develop outside the body, but the life history is not known. The parasite occupies the upper portion of the small intestine. Usually not more than one or two are present, but occasionally they occur in enormous numbers. The migrations are peculiar. They may pass into the stomach, from which they may be ejected by vomiting, or they may crawl up the œsophagus and enter the pharynx, from which they may be withdrawn. A child, under my care in the small-pox department of the General Hospital, during convalescence, withdrew in this way more than thirty round worms within a few weeks. In other instances the worm passes into the larynx, and has been known to cause fatal asphyxia, or passing into the trachea, to cause gangrene of the lung. They may pass into the Eustachian tube and appear at the external meatus. The most serious migration is into the bile-duct. There is a specimen in the Wistar-Horner Museum of the University of Pennsylvania in which not only the common duct, but also the main branches throughout the liver are enormously distended and packed with numerous round worms. The bowel may be perforated by them and peritonitis result.

The symptoms are not definite. When a few are present they may be passed without causing disturbance. In children there are irritative symptoms usually attributed to worms, such as restlessness, irritability, picking at the nose, grinding of the teeth, twitchings, or convulsions. These symptoms may be marked in very nervous children.

Treatment.—Santonin can be given, mixed with sugar, in doses of from one to three grains for a child and three to five grains for an adult, followed by a calomel or a saline purge. The dose may be given for

three or four days. An unpleasant consequence which sometimes follows the administration of this drug is xanthopsia or yellow vision.

(*b*) *Oxyuris Vermicularis* (*Thread-worm; Pin-worm*).—This common parasite occupies the rectum and colon. The male measures about four millimetres in length, the female about ten millimetres. They produce great irritation and itching, particularly at night, symptoms which become intensely aggravated by the nocturnal migration of the parasites.

The patients become extremely restless and irritable, the sleep is often disturbed, and there may be loss of appetite and anæmia. Though most common in children the parasite occurs at all ages.

The worm is readily detected in the fæces. Infection probably takes place through the water or possibly through salads, such as lettuces and cresses. A person the subject of the worms passes ova in large numbers in the fæces, and the possibility of reinfection must be scrupulously guarded against.

The treatment is simple, though occasionally there are instances in which all forms of medication are resisted. A case is mentioned of a gentleman, aged forty, who had suffered from childhood and had failed to obtain any benefit from prolonged treatment by many helminthologists. Santonin may be used in small doses, and mild purgatives, particularly rhubarb. Large injections containing carbolic acid, vinegar, quassia, aloes, or turpentine may be employed. In children the use of cold injections of strong salt and water is usually efficacious. They should be repeated for at least ten days. In giving the injection care should be taken to have the hips well elevated so that the fluid can be retained as long as possible. For the intense itching and irritation at night vaseline may be freely used or belladonna ointment.

II. Trichiniasis.

The trichina spiralis in its adult condition lives in the small intestine. The disease is produced by the embryos, which pass from the intestines and reach the voluntary muscles, where they finally become encapsulated —muscle trichinæ. It is in the migration of the embryos that the group of symptoms known as trichiniasis is produced.

Description of the Parasites.—(*a*) Adult or intestinal form. The female measures from three to four millimetres; the male, 1·5 millimetre, and has two little projections from the hinder end.

(*b*) The embryo or muscle trichina is from 0·6 to one millimetre in length and lies coiled in an ovoid capsule, which is at first translucent, but subsequently opaque and infiltrated with lime salts. The worm presents a pointed head and a somewhat rounded tail.

When flesh containing the trichinæ is eaten by man or by any animal in which the development can take place, the capsules are digested and the trichinæ set free. They pass into the small intestine, and about

the third day attain their full growth and become sexually mature. Virchow's experiments have shown that on the sixth or seventh day the embryos are fully developed. The young produced by each female trichina have been estimated at several hundred. Leukart thinks that various broods are developed in succession, and that as many as a thousand embryos may be produced by a single worm. The time from the ingestion of the flesh containing the muscle trichinæ to the development of the brood of embryos in the intestines is from seven to nine days. As soon as born the embryo trichinæ leave the intestines; wandering through the peritonæum and the connective tissues, probably through the mesentery and retroperitoneal tissues—some hold by means of the blood current—they finally reach the muscles, which constitute "the seat of election." After a preliminary migration in the intermuscular connective tissue they penetrate the primitive muscle fibres, and in about two weeks develop into the full-grown muscle form. In this process an interstitial myositis is excited and gradually an ovoid capsule develops about the parasite. Two, occasionally three or four, worms may be seen within a single capsule. This process of encapsulation has been estimated to take about six weeks. Within the muscles the parasites do not undergo further development. Gradually the capsule becomes thicker, and ultimately lime salts are deposited within it. This change may take place in man within four or five months. In the hog it may be deferred for many years. The calcification renders the cyst visible, and since first seen by Tiedemann, in 1822, and Hilton, in 1832, these small, opaque, oat-shaped bodies have been familiar objects to demonstrators of normal and morbid anatomy. The trichinæ may live within the muscles for an indefinite period. They have been found alive and capable of developing as late as twenty or even twenty-five years after their entrance into the system. In many instances, however, the worms are completely calcified. The trichina occurs in swine, in the rat, occasionally in mice and cats; it has been found also in the fox and a few other animals. The parasite was first found in the hog by the late Joseph Leidy. Experimentally, guinea-pigs and rabbits are readily infected by feeding them with muscle containing the larval form. Dogs are infected with difficulty; cats more readily. Experimentally, animals sometimes die of the disease if large numbers of the parasites have been eaten. In the hog the trichinæ, like the cysticerci, cause few if any symptoms. An animal the muscles of which are swarming with living trichinæ may be well nourished and healthy-looking. An important point also is the fact that in the hog the capsule does not readily become calcified, so that the parasites are not visible as in the human muscles. For a long time the trichina was looked upon as a pathological curiosity, but in 1860 Zenker discovered in a girl in the Dresden Hospital who had symptoms of typhoid fever both the intestinal and the muscle forms of the trichinæ, since which time the disease has been thoroughly studied.

Man is infected by eating the flesh of trichinous hogs. The incidence of the disease in swine varies much in different countries. In Germany, where a thorough and systematic microscopic examination of all swine flesh is made, the proportion of trichinous hogs is about 1 in 1,852. At the Berlin abattoir, where the microscopic examination is conducted by a staff of over eighty men and women, two portions are taken from the abdominal muscles, from the diaphragm, and from the intercostal muscles, and one piece from the muscles of the larynx and tongue. A special compressor is used to flatten the fragments of the muscle, and the examination is made with a magnifying power of from seventy to one hundred diameters. During the three years ending in 1885 there were 603 trichinous hogs detected, a ratio of 1 to 1,292. Statistics are not available in England. In the United States systematic inspection is unknown, and the statistics are by no means extensive enough. "Taking all the examinations of American pork thus far made, both at home and abroad, and we have a total of 298,782, in which trichinæ were found 6,280 times, being 2·1 per cent, or 1 to 48" (Salmon, 1884).

In 1883, in conjunction with A. W. Clement, I examined 1,000 hogs at the Montreal abattoir, and found only 4 infected. There is no reason to believe that the hog of this country is less liable to trichina than the German animal.

Modes of Infection.—The danger of infection depends entirely upon the mode of preparation of the flesh. Thorough cooking, so that all parts of the meat reach the boiling point, destroys the parasites; but in large joints the central portions are often not raised to this temperature. The frequency of the disease in different countries depends largely upon the habits of the people in the preparation of pork. In North Germany, where raw ham and *wurst* are freely eaten, the greatest number of cases have occurred. In South Germany, France, and England cases are rare. In this country the greatest number of persons attacked have been Germans. Salting and smoking the flesh are not always sufficient, and the Havre experiments showed that animals are readily infected when fed with portions of the pickled or the smoked meat as prepared in this country. Carl Fränkel, however, states that the experiments on this point have been negative, and that it is very doubtful if any cases of trichiniasis in Germany have been caused by American pork.

Frequency of Infection.—The dissecting-room and post-mortem statistics show that from one half to two per cent of all bodies contain trichinæ. Of 1,000 consecutive autopsies of which I have notes the trichinæ were present in 6 instances. I have, in addition, seen them in two dissecting-room cases and in two bodies at the Philadelphia Hospital.

The disease often occurs in epidemics, a large number of persons being infected from a single source. Among the best known of these outbreaks are the Hedersleben, in which there were 337 persons affected, and the Emersleben, in which there were 250 persons attacked. The extensive

outbreaks of this sort have been, with few exceptions, in North Germany. Alfred Mann, after a careful search, at my request, of the literature in the Surgeon-General's library, finds records of 456 cases in this country. The two largest groups of cases were at Astoria, Ore., reported by Kinney, 15 cases and one death; and at St. Paul, Minn., reported by Persons and Andrews, 15 cases and three deaths.

Symptoms.—The ingestion of trichinous flesh is not necessarily followed by the disease. When a limited number are eaten only a few embryos pass to the muscles and may cause no symptoms. Well-characterized cases present a gastro-intestinal period and a period of general infection.

In the course of a few days after eating the infected meat there are signs of gastro-intestinal disturbance—pain in the abdomen, loss of appetite, vomiting, and sometimes diarrhœa. The preliminary symptoms, however, are by no means constant, and in some of the large epidemics cases have been observed in which they have been absent. In other cases the gastro-intestinal features have been marked from the outset, and the attack has resembled cholera nostras. Pains in different parts of the body, general debility, and weakness have been noted in some of the epidemics.

The invasion symptoms develop between the seventh and the tenth day, sometimes not until the end of the second week. There is fever, except in very mild cases Chills are not common. The thermometer may register 102° or 104°, and the fever is usually remittent or intermittent. The migration of the parasites in the muscles excites a more or less intense myositis, which is characterized by pain on pressure and movement, and by swelling and tension of the muscles. The limbs are placed in the positions in which the muscles are in least tension. The involvement of the muscles of mastication and of the larynx may cause difficulty in chewing and swallowing. In severe cases the involvement of the diaphragm and intercostal muscles may lead to intense dyspnœa, which sometimes proves fatal. Œdema, a feature of great importance, may be early in the face. Later it develops in the extremities when the swelling and stiffness of the muscles are at their height. Profuse sweats, tingling and itching of the skin, and in some instances urticaria, have been described. The general nutrition is much disturbed and the patient becomes emaciated and often anæmic, particularly in the protracted cases. The patellar tendon reflex may be absent. The patients are usually conscious, except in cases of very intense infection, in which the delirium, dry tongue, and tremors give a picture similar to typhoid fever. In addition to the dyspnœa, present in the severer cases, there may be bronchitis, and in the fatal cases pneumonia or pleurisy. In some epidemics polyuria has been a common symptom. Albuminuria is frequent.

The intensity and duration of the symptoms depend entirely upon the grade of infection. In the mild cases recovery is complete in from ten to fourteen days. In the severe forms convalescence is not established for six or eight weeks, and it may be months before the patient recovers the

muscular strength. One case in the Hedersleben epidemic was weak eight years after the attack.

Of 72 fatal cases in the Hedersleben epidemic the greatest mortality occurred in the fourth and fifth and sixth weeks; namely, 52 cases. Two died in the second week with severe choleraic symptoms.

The mortality has ranged in different outbreaks from one or two per cent to thirty per cent. In the Hedersleben epidemic 101 persons died. Among the 456 cases reported in this country there were 122 deaths.

The *anatomical changes* are chiefly in the voluntary muscles. In the early stages they look normal, but in the fourth or fifth week grayish-white areas appear in which the muscle fibres are extensively degenerated and in the neighborhood of the trichinæ there is an acute interstitial myositis. Cohnheim has described a fatty degeneration of the liver and enlargement of the mesenteric glands. At the time of death in the fourth or fifth week or later the adult trichinæ are still found in the intestines.

The *prognosis* depends much upon the quantity of infected meat which has been eaten and the number of trichinæ which mature in the intestines. In children the outlook is more favorable. Early diarrhœa and moderately intense gastro-intestinal symptoms are, as a rule, more favorable than constipation.

Diagnosis.—This is perfectly clear when a large number of persons are infected at once and the parasites have been found in the ham or sausages. The worms may be discovered in the stools. The stools should be spread on a glass plate or black background and examined with a low-power lens, when the trichinæ are seen as small, glistening, silvery threads. In doubtful cases the diagnosis may be made by the removal of a small fragment of muscle. A special harpoon has been devised for this purpose by means of which a small portion of the biceps or of the pectoral muscle may be readily removed. Under cocaine anæsthesia an incision may be made and a small fragment removed. The disease may be mistaken for acute rheumatism, particularly as the pains are so severe on movement, but there is no special swelling of the joints. The tenderness is in the muscles both on pressure and on movement. The intensity of the gastro-intestinal symptoms in some cases has led to the diagnosis of cholera. Many of the former epidemics were doubtless described as typhoid fever, which the severer cases, owing to the prolonged fever, the sweats, the delirium, dry tongue, and gastro-intestinal symptoms, somewhat resemble. The pains in the muscles, swelling, œdema, and shortness of breath are the most important diagnostic points. Under acute myositis reference has already been made to the cases which closely resemble trichiniasis. The epidemic in 1879 on board the training ship Cornwall presented symptoms similar to those of trichiniasis. One patient died. Two months after burial the body was examined, and living and dead nematode worms were found which, as Bastian showed, were not the trichina, but a rhabditis.

and in the Sandwich Islands (Lutz).

They were probably not parasitic, but entered the body of the cadet after burial.

Prophylaxis.—It is not definitely known how swine become diseased. It has been thought that they are infected from rats about slaughter-houses, but it is just as reasonable to believe that the rats are infected by eating portions of the trichinous flesh of swine. The swine should, as far as possible, be grain-fed, and not, as is so common, allowed to eat offal. The most satisfactory prophylaxis is the complete cooking of pork and sausages, and to this custom in England, France, South Germany, and particularly in this country, immunity is largely due.

Treatment.—If it has been discovered within twenty-four or thirty-six hours that a large number of persons have eaten infected meat, the indications are to thoroughly evacuate the gastro-intestinal canal. Purgatives of rhubarb and senna may be given, or an occasional dose of calomel. Glycerin has been recommended in large doses in order that by passing into the intestines it may by its hygroscopic properties destroy the worm. Male-fern, kamala, santonin, and thymol have all been recommended in this stage. There is no doubt that diarrhœa in the first week or ten days of the infection is distinctly favorable. The indications in the stage of invasion are to relieve the pains, to secure sleep, and to support the patient's strength. There are no medicines which have any influence upon the embryos in their migration through the muscles.

III. Anchylostomiasis.

The *dochmius* or *strongylus duodenalis*, also known as the *sclerostomum* or *anchylostomum duodenale* is the only strongyle harmful to man. It belongs to the same family as the *strongylus armatus*, which causes the verminous aneurism in the horse. The parasites live in the upper portion of the small intestine, chiefly in the jejunum. They are easily seen, the male having a length of from six to ten millimetres, and the female from ten to eighteen millimetres. The mouth is provided with a series of tooth-like hooks, by means of which the parasite attaches itself to the mucous membrane. The male has a prominent expansion or bursa at the tail end. The existence of the parasite has long been known, but it was not thought to be pathogenic until Griesinger demonstrated its association with the Egyptian chlorosis. It has also been shown to be the cause of the anæmia to which miners and brick-makers are subject. Throughout Europe the disease has been widely spread by the employment of Italian and Polish laborers. In certain Italian provinces it is extremely prevalent and serious. It occurs in India and in Brazil, and has been described in Jamaica (Strachan). Dolley states that the parasite was described many years ago by physicians in the Southern States, but no recent observations upon the disease have been made in this country.

Symptoms.—The parasites withdraw blood by suction, and the

symptoms result from this slow depletion. In the early stage there may only be gastric or gastro-intestinal disturbance, but if the parasites are present in large numbers anæmia is gradually produced and constitutes the characteristic feature of the disease. The Egyptian chlorosis, brick-maker's anæmia, tunnel anæmia, miner's cachexia, and mountain anæmia are due to this cause. The clinical course is variable. In some instances the anæmia develops acutely and reaches a high grade within a short time, causing great shortness of breath and œdema. There is serious disturbance of nutrition, sometimes diarrhœa and colicky pains; but the most pronounced symptom is the pallor and the associated phenomena of chronic anæmia. The lesions of the intestines are those of chronic catarrh, and small hæmorrhages occur in the mucosa. Dilatation and hypertrophy of the heart have been found in many cases.

The diagnosis is not difficult. The ova, which are abundant in the stools, are oval, about ·05 millimetre in length, and possess a thin, transparent shell. There is no operculum, as in the ovum of the oxyuris, and the yolk is unsegmented. The larvæ develop in moist earth and readily get into the drinking water, through which infection occurs.

The systematic use of latrines and the boiling of all water used for drinking purposes are the important prophylactic measures. The treatment should be directed to the destruction of the parasites in the intestine, which may be effected by the male fern or by thymol, which Sonsino recommends highly. It is given in capsules of half a drachm every hour for four doses. A purgative is not necessary.

IV. Filariasis.

Under this term may be considered the morbid conditions induced by the *filaria sanguinis hominis*, or the *filaria Bancrofti*, the name employed to designate the adult worm, which was discovered by Bancroft, of Brisbane. In the adult form the worm lives in the lymphatics. The female is thus described by Patrick Manson, whose studies on this parasite have been so important: "A long, slender, hair-like animal quite three inches in length but only one one hundredth inch in breadth, of an opaline appearance, looking, as it lies in the tissues, like a delicate thread of catgut animated and wriggling. A narrow alimentary canal runs from the simple club-like head to within a short distance of the tail, the remainder of the body being almost entirely occupied by the reproductive organs. The vagina opens about one twenty-fifth of an inch from the head; it is very short, and bifurcates into two uterine horns, which, stuffed with embryos in all stages of development, run backward nearly to the tail." The male worm is much smaller and has only occasionally been found. The female produces an extraordinary number of embryos, which enter the blood current through the lymphatics. Each embryo is within its shell, which is elongated, scarcely perceptible, and in no way impedes the movements.

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monog of Giles.

Anæmia

The worms are found within 2 metres of the pylorus, often with their heads buried in the mucosa but Splen consolidation.

Sandwith, ~~has~~ relates that in Egypt. the disease is most frequently met with in men nearly all in peasants who work in the damp earth — in many of whom are Earth eaters.

Thymol recommended by Bozzolo in 1888 is a specific and should be given in large doses, viz. 2 grammes at 8 am (in water) & 2 grammes at 10 am given in pill to patients with brandy (Sandwith). The diet should be Milk & soup. Two hours after the second dose of thymol a purge of castor oil or magnesia is given The patient may still require a second dose but if necessary it may be repeated in a week

They are about the ninetieth part of an inch in length and the diameter of a red blood-corpuscle in thickness, so that they readily pass through the capillaries. They move with the greatest activity and form very striking and readily recognized objects in a blood-drop under the microscope. A remarkable feature is the periodicity in the occurrence of the embryos in the blood. In the daytime they are almost or entirely absent, whereas at night, in typical cases, they are present in large numbers. If, however, as Stephen Mackenzie has shown, the patient, reversing his habits, sleeps during the day, the periodicity is reversed. The further development of the embryos appears to be associated with the mosquito, which at night sucks the blood and in this way frees them from the body. Some slight development takes place within the body of the mosquito, and it is probable that the embryos are set free in the water after the death of the host. The further development is not known, but it is probably in drinking water. The filariæ may be present in the body without causing any symptoms. In animals blood filariæ are very common and rarely cause inconvenience. It is only when the adult worms or the ova block the lymph channels that certain definite symptoms occur. Manson suggests that it is the ova (prematurely discharged), which are considerably shorter and thicker than the full-grown embryos, which block the lymph channels and produce the conditions of hæmatochyluria, elephantiasis, and lymph-scrotum.

The parasite is widely distributed, particularly in tropical and subtropical countries. Guitéras has shown that the disease prevails extensively in the Southern States, and since his paper appeared contributions have been made by Matas, of New Orleans, Mastin, of Mobile, and De Saussure, of Charleston.

The effects produced may be described under the above-mentioned conditions.

(*a*) *Hæmatochyluria.*—Without any external manifestations, and in many cases without special disturbance of health, the subject from time to time passes urine of an opaque white, milky appearance, or bloody, or a chylous fluid which on settling shows a slightly reddish clot. The urine may be normal in quantity or increased. The condition is usually intermittent, and the patient may pass normal urine for weeks or months at a time. Microscopically, the chylous urine contains minute molecular fat granules, usually red blood-corpuscles in various amounts. It was in urine of this kind that Wucherer, of Bahia, first detected the filarian embryos. It is remarkable for how long the condition may persist without serious impairment of the health. A patient, sent to me by Dawson, of Charleston, has had hæmatochyluria intermittently for eighteen years. The only inconvenience has been in the passage of the blood-clots which collect in the bladder. At times he has also uneasy sensations in the lumbar region. The embryos are present in his blood at night in large numbers. Chyluria is not always due to the filaria. The non-parasitic form of the disease has already been considered.

Opportunities for studying the anatomical condition of these cases rarely occur. In the case described by Stephen Mackenzie the renal and peritoneal lymph plexuses were enormously enlarged, extending from the diaphragm to the pelvis. The thoracic duct above the diaphragm was impervious.

(*b*) *Lymph-scrotum* and certain forms of *elephantiasis* are sometimes caused by the filaria. In the former the tissues of the scrotum are enormously thickened and the distended lymph-vessels may be plainly seen. A clear, sometimes a turbid, fluid follows puncture of the skin. The parasites are not always to be found. I have examined two typical cases without finding filaria in the exuded fluids or in the blood at night. So also the majority of cases of elephantiasis which occur in this country are non-parasitic. In China it is stated that the parasites occur in all these cases.*

V. Dracontiasis (*Guinea-worm Disease*).

The *Filaria* or *Dracunculus medinensis* is a widely spread parasite in parts of Africa and the East Indies. In the United States cases occasionally occur. Jarvis reports a case in a post chaplain who had lived at Fortress Monroe, Va., for thirty years. Van Harlingen's patient, a man aged forty-seven, had never lived out of Philadelphia, so that the worm must be included among the parasites of this country. A majority of the cases reported in American journals have been imported.

Only the female is known. It develops in the subcutaneous and intermuscular connective tissues and produces vesicles and abscesses. In the large majority of the cases the parasite is found in the leg. Of 181 cases, in 124 the worm was found in the feet, 33 times in the leg, and 11 times in the thigh. The worm is usually solitary, though there are cases on record in which six or more have been present. It is cylindrical in form, about two millimetres in diameter, and from fifty to eighty centimetres in length.

The worm gains entrance to the system through the stomach, not through the skin, as was formerly supposed. It is probable that both male and female are ingested; but the former dies and is discharged, while the latter after impregnation penetrates the intestine and attains its full development in the subcutaneous tissues, where it may remain quiescent for a long time and can be felt beneath the skin like a bundle of string. Suppuration is after a time excited, and when the abscesses are opened or burst the worm appears and is sometimes discharged entire. The worm contains an enormous number of living embryos, which escape into the water and develop in the cyclops—a small crustacean—and it seems likely that man is infected by drinking the water containing these developed larvæ.

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* For full consideration of the subject of congenital occlusion and dilatation of lymph channels, see work on this subject by Samuel C. Busey, New York, 1878.

The *treatment* consists in promoting the suppuration, and when the worm is seen the common procedure is to roll it round a portion of smooth wood, and in this way prevent the retraction, and each day wind a little more until the entire worm is withdrawn. It is stated that special care must be taken to prevent tearing of the worm, as disastrous consequences sometimes follow, probably from the irritation caused by the migration of the embryos. It is stated that the leaves of the plant called *amarpattee* are almost a specific in the disease. Asafœtida in full doses is said to kill the worm.

VI. Other Nematodes.

(*a*) Among less important filarian worms parasitic in man the following may be mentioned: *filaria loa*, which is a cylindrical worm of about three centimetres in length and whose habitat is beneath the conjunctiva. It has been found on the West African coast, in Brazil, and in the West Indies. *Filaria lentis*, which has been found in a cataract. Three specimens have been found together. *Filaria labialis*, which has been found in a pustule in the upper lip *Filaria hominis oris*, which was described by Leidy, from the mouth of a child. *Filaria bronchialis*, which has been found occasionally in the trachea and bronchi. This parasite has been seen in a few cases in the bronchioles and in the lungs. There is no evidence that it ever produces an extensive verminous bronchitis similar to that which I have described in dogs. *Filaria imitis*, of which Bowlby has described two cases. In one case with hæmaturia female worms were found in the portal vein, and the ova were present in the thickened bladder wall and in the ureters.

(*b*) *Trichocephalus Dispar* (*Whip-worm*).—This parasite is not infrequently found in the cæcum and large intestine of man. It measures from four to five centimetres in length, the male being somewhat shorter than the female The worm is readily recognized by the remarkable difference between the anterior and posterior portions. The former, which is at least three fifths of the body, is extremely thin and hair-like in contrast to the thick hinder portion of the body, which in the female is conical and pointed, and in the male more obtuse and usually rolled like a spring. The ova are oval, lemon-shaped, ·05 millimetre in length, and each is provided with a button-like projection.

The number of the worms found is variable, as many as a thousand having been counted. It is a widely spread parasite. In parts of Europe it occurs in from ten to thirty per cent of all bodies examined, but in this country it is not so common. The trichocephalus rarely causes symptoms. It has been thought by certain physicians in the East to be the cause of beri-beri. Several cases have been reported recently in which profound anæmia has occurred in connection with this parasite, usually with diarrhœa. Enormous numbers may occur, as in Rudolphi's case, without producing any symptoms.

The diagnosis is readily made by the examination of the fæces, which contain, sometimes in great abundance, the characteristic lemon-shaped, hard, dark-brown eggs.

(*c*) *Eustrongylus Gigas.*—This enormous nematode, the male of which measures about a foot in length and the female about three feet, occurs in very many animals and has occasionally been met with in man. It is usually found in the renal region and may entirely destroy the kidney.

(*d*) *Rhabdonema Intestinale.*—Under this name are now included the small nematode worms found in the fæces and formerly described as *anguillula stercoralis* and *anguillula intestinalis.* This parasite occurs abundantly in the stools of the endemic diarrhœa of hot countries, and has been specially described by the French in the diarrhœa of Cochin-China. It occurs also in Brazil, and has been found in Italy in connection with the anchylostoma in cases of miner's anæmia. It is stated that the worms occupy all parts of the intestines, and have even been found in the biliary and pancreatic ducts. It is only when they are in very large numbers that they produce severe diarrhœa and anæmia.

Acanthocephala (*Thorn-headed Worms*).—The *echinorhynchus gigas* is a common parasite in the intestine of the hog and attains a large size. The larvæ develop in cockchafer grubs. Lambl found a small *echinorhynchus* in the intestine of a boy. Welch's specimen, which was found encysted in the intestine of a soldier at Netley, is stated by Cobbold probably not to have been an *echinorhynchus.* Recently a case of *echinorhynchus moniliformis* has been described in Italy by Grassi and Calandruccio.

IV. DISEASES CAUSED BY CESTODES

(*Tape-worms; Hydatid Disease*).

Man harbors the adult parasites in the small intestine, the larval forms in the muscles and solid organs.

I. Intestinal Cestodes; Tape-worms.

(*a*) *Tænia solium*, or pork tape-worm. This is not a common form in this country. It is much more frequent in parts of Europe and Asia. When mature it is from six to twelve feet in length. The head is small, round, not so large as the head of a pin, and provided with four sucking disks and a double row of hooklets; hence it is called, in contradistinction to the other form in man, the armed tape-worm. To the head succeeds a narrow, thread-like neck, then the segments, or proglottides, as they are called. The segments possess both male and female generative organs, and about the four hundred and fiftieth become mature and contain ripe ova. The worm attains its full growth in from three to three and a half months, after which time the segments are continuously shed and appear

in the stools. The segments are about one centimetre in length and from seven to eight millimetres in breadth. Pressed between glass plates the ovarian rosette is seen as a central stem with about twelve or fifteen lateral branches. There are many thousands of ova in each ripe segment, and each ovum consists of a firm shell, inside of which is a little embryo, provided with six hooklets. The segments are continuously passed, and if the ova are to attain further development they must be taken into the stomach, either of a pig, or of man himself. The egg-shells are digested, the six-hooked embryos become free, and passing from the stomach reach various parts of the body (the liver, muscles, brain, or eye), where they develop into the larvæ or cysticerci. A hog under these circumstances is said to be *measled*, and the cysticerci are spoken of as measles or bladder worms.

The *tænia solium* received its name because it was thought to exist as a solitary parasite in the bowel, but two or three, or even more worms may occur.

(*b*) *Tænia saginata* or *mediocanellata*—the unarmed or beef tape-worm. This is a longer and larger parasite than the *tænia solium*. It is certainly the common tape-worm of this country. Of scores of specimens which I have examined, almost all were of this variety. According to Bérenger-Féraud it has spread rapidly in western Europe, owing probably to the importation of beef and live stock from the Mediterranean basin. It may attain a length of fifteen or twenty feet, or more. The head is large in comparison to the *tænia solium*, and measures over two millimetres in breadth. It is square-shaped and provided with four large sucking disks, but there are no hooklets. The ripe segments are from seventeen to eighteen millimetres in length, and from eight to ten millimetres in breadth. The ovarian rosette consists of a central stem with from seventeen to eighteen lateral branches, which are given off more dichotomously than in the *tænia solium*. The ova are somewhat larger, and the shell is thicker, but the two forms can scarcely be distinguished by their ova. The ripe segments are passed as in the tænia solium, and are ingested by cattle, in the flesh or organs of which the eggs develop into the bladder worms or cysticerci. Whether they develop in man or not is uncertain. No instance of the cysticercus of the *tænia saginata* has, so far as I know, been reported in man.

Of other forms of tape-worm may be mentioned:

(*c*) *Tænia elliptica* (*tænia cucumerina*). A small parasite very common in the dog and occasionally found in man, and the larvæ of which develop in the louse of the dog.

(*d*) *Tænia flavo-punctata*. A small cestode was found in the intestine of a child in Boston, and has since been met with in one or two cases.

(*e*) *Tænia nana* and the *tænia Madagascariensis* have been found only once or twice.

(*f*) *Bothriocephalus latus.* A cestode worm found only in certain districts bordering on the Baltic Sea and in parts of Switzerland. So far as I know it has not been found in this country except in a few imported cases. The parasite is large and long, measuring from twenty-five to thirty feet or more. Its head is different from that of the tænia, as it possesses two lateral grooves or pits and has no hooklets. The larvæ develop in the peritonæum and muscles of the pike and other fish, and it has been shown experimentally that they grow into the adult worm when eaten by man.

Symptoms.—These parasites are found at all ages. They are not uncommon in children and are occasionally found in sucklings. W. T. Plant refers to a number of cases in children under two years, and there is a case in the literature in which it is stated that the tape-worm was found in an infant five days old.

The parasites may cause no disturbance and are rarely dangerous. A knowledge of the existence of the worm is generally a source of worry and anxiety; the patient may have considerable distress and complain of abdominal pains, nausea, and sometimes diarrhœa. Occasionally, the appetite is ravenous. In women and in nervous patients the constitutional disturbance may be considerable, and we not infrequently see great mental depression and even hypochondria. Various nervous phenomena, such as chorea, convulsions, or epilepsy, are believed to be caused by the parasites. Such effects, however, are very rare.

The *diagnosis* is never doubtful. The presence of the segments is distinctive. The ova, too, may be recognized in the stools. It makes but little difference as to the form of tape-worm, but the ripe segments of the *tænia saginata* are larger and broader, and show differences in the generative system as already mentioned.

The *prophylaxis* is most important. Careful attention should be given to two points. First, all tape-worm segments should be burned. They should never be thrown into the water-closet or outside. And second, the meat should be cooked throughout, in which way alone larvæ are destroyed. Possibly it is owing to the fact that in this country pork is, as a rule, better cooked than beef that the *tænia saginata* is the most common form. Certainly in the market and at the abattoirs one more commonly sees measly pork than measly veal. In the examination of a thousand hogs in Montreal there were seventy-six instances of cysticerci. The measle is more readily overlooked in beef than in pork, as in the former it has not such an opaque white color.

Treatment.—For two days prior to the administration of the remedies the patient should take a very light diet and have the bowels moved occasionally by a saline cathartic. The practitioner has the choice of a large number of drugs. As a rule, the male fern acts promptly and well. The ethereal extract, in two-drachm doses, may be given fasting, and followed in the course of a couple of hours by a brisk purgative. This usu-

ally succeeds in bringing away a large portion, but not always the entire worm.

A combination of the remedies is sometimes very effective. An infusion is made of pomegranate root, half an ounce; pumpkin seeds, one ounce; powdered ergot, a drachm; and boiling water, ten ounces. To an emulsion of the male fern (a drachm of ethereal extract), made with acacia powder, two minims of croton oil are added. The patient should have had a low diet the previous day and have taken a dose of salts in the evening. The emulsion and infusion are mixed and taken fasting at nine in the morning.

The pomegranate root is a very efficient remedy, and may be given as an infusion of the bark, three ounces of which may be macerated in ten ounces of water and then reduced to one half by evaporation. The entire quantity is then taken in divided doses. It occasionally produces colic, but is a very effective remedy. The active principle of the root, pelletierine, is now much employed. It is given in doses of one fourth to one half of a grain, and is followed in an hour by a purge.

Pumpkin seeds are sometimes very efficient. Three or four ounces should be carefully bruised and then macerated for twelve or fourteen hours and the entire quantity taken and followed in an hour by a purge. Of other remedies, koosso, turpentine in ounce doses in honey, and kamala may be mentioned.

Unless the head is brought away, the parasite continues to grow, and within a few months the segments again appear. Some instances are extraordinarily obstinate. Doubtless it depends a good deal upon the exposure of the worm. The head and neck may be thoroughly protected beneath the valvulæ conniventes, in which case the remedies may not act. Owing to its armature the *tænia solium* is more difficult to expel. It is probable that no degree of peristalsis could dislodge the head, and unless the worm is killed it does not let go its extraordinarily firm hold on the mucous membrane.

II. Visceral Cestodes.

Whereas adult tæniæ cause little or no disturbance and rarely, if ever, prove directly fatal, the affections caused by the larvæ or immature forms in the solid organs are serious and important. There are two chief cestode larvæ known to frequent man—(*a*) the *cysticercus cellulosæ*, the larva of the *tænia solium*, and (*b*) the *echinococcus*, the larva of the *tænia echinococcus*.

I. **Cysticercus Cellulosæ.**—When man accidentally takes into his stomach the ripe ova of *tænia solium* he is liable to become the intermediate host, a part usually played for this tape-worm by the pig. This accident may occur in an individual the subject of *tænia solium*, in which case the mature proglottides either themselves wander into the stomach or, what is more likely, are forced into the organ in attacks of prolonged

vomiting. Of course the accidental ingestion from the outside of a few ova is quite possible, and the liability of infection should always be borne in mind in handling the segments of the worm.

The symptoms depend entirely upon the number of ova ingested and the localities reached. In the hog the cysticerci produce very little disturbance. The muscles, the connective tissue, and the brain may be swarming with the measles, as they are called, and yet the nutrition is maintained and the animal does not appear to be seriously incommoded. In the invasion period, if large numbers of the parasites are taken, there is, in all probability, constitutional disturbance; certainly there is in the calf, when fed with the ripe segments of *tænia saginata*.

In man a few cysticerci lodged beneath the skin or in the muscles may cause no damage, and in time the larvæ die and become calcified. They are occasionally found in dissection subjects or in post-mortems as ovoid white bodies in the muscles or subcutaneous tissue. In this country they are very rare. I have seen but one instance in my post-mortem experience. Depending on the number and the locality specially affected, the symptoms may be grouped into general, cerebro-spinal, and ocular.

(1) *General.*—As a rule the invasion of the larvæ in man, unless in very large numbers, does not cause very definite symptoms. It occasionally happens, however, that a striking picture is produced. For instance, a patient was admitted to my wards very stiff and helpless, so much so that he had to be assisted up-stairs and into bed. He complained of numbness and tingling in the extremities and general weakness, so that at first he was thought to have a peripheral neuritis. At the examination, however, a number of painful subcutaneous nodules were discovered, which proved on excision to be the cysticerci. Altogether seventy-five could be felt subcutaneously, and from the soreness and stiffness they probably existed in large numbers in the muscles. There were none in his eyes, and he had no symptoms pointing to brain lesions.

(2) *Cerebro-spinal.*—Remarkable symptoms may result from the presence of the cysticerci in the brain and cord. In the silent region they may be abundant without producing any symptoms. I have in my possession the brain of a pig containing scores of "measles," yet the animal in the few moments in which I saw it just prior to death did not present any symptoms to attract attention. In the ventricles of the brain the cysticerci may attain a considerable size, owing to the fact that in regions in which they are unrestrained in their growth the bladder-like body grows freely, as in the peritonæum. When in the fourth ventricle remarkable irritative symptoms may be produced. In 1884 I saw with Friedländer in Berlin a case from Riess's wards in which during life there had been symptoms of diabetes and anomalous nervous symptoms. Post mortem, the cysticercus was found beneath the valve of Vieussens, pressing upon the floor of the left ventricle.

(3) *Ocular.*—Since von Graefe demonstrated the presence of the cysti-

fourth

cercus in the vitreous humor many cases have been placed on record, and it is a condition easily recognized by oculists.

Except in the eye, the diagnosis can rarely be made; when the cysticerci are subcutaneous, one may be excised. It is possible that when numerous throughout the muscles they may be seen under the tongue, in which situation they may exist in the pig in numbers.

II. **Echinococcus Disease.**—The hydatid worms or echinococci are the larvæ of the *tænia echinococcus* of the dog. This is a tiny cestode not more than four or five millimetres in length, consisting of only three or four segments, of which the terminal one alone is mature, and has a length of about two millimetres and a breadth of 0·6 millimetre. The head is small and provided with four sucking disks and a rostellum with a double row of hooklets. This is an exceedingly rare parasite in the dog. Cobbold states that he has never met with a natural specimen in England. Leidy had not one in his large collection. I have not met with an instance in this country, nor do I know of its ever having been described. The only specimens in my cabinet I procured experimentally by feeding a dog with echinococcus cysts from an ox. The worms are so small that they may be readily overlooked, since they form small white, thread-like bodies closely adherent among the villi of the small intestines. The ripe segment contains about 5,000 eggs, which attain their development in the solid organs of various animals, particularly the hog and ox; more rarely the horse and the sheep. In some countries man is a common intermediate host, owing to the accidental ingestion of the ova.

Development.—The little six-hooked embryo, freed from the egg-shell by digestion, ~~either~~ burrows through the intestinal wall and reaches the peritoneal cavity or the muscles; ~~more commonly~~ it enters the portal vessels and is carried to the liver. It may enter the systemic vessels, and, passing the pulmonary capillaries, as it is protoplasmic and elastic, may reach the brain or other parts. Once having reached its destination, it undergoes the following changes: The hooklets disappear and the little embryo is gradually converted into a small cyst which presents two distinct layers—an external, laminated, cuticular membrane or capsule, and an internal, granular, parenchymatous layer, the endocyst. The little cyst or vesicle contains a clear fluid. There is more or less reaction in the neighboring tissues, and the cyst in time has a fibrous investment. When this primary cyst or vesicle has attained a certain size buds develop from the parenchymatous layer, which are gradually converted into cysts, presenting a structure identical with that of the original cyst, namely, an elastic chitinous membrane lined with a granular parenchymatous layer. These secondary or daughter cysts are first connected with the lining membrane of the primary, but are soon set free. In this way the primary cyst as it grows may contain a dozen or more daughter cysts. Inside these daughter cysts a similar process may occur, and from buds in the walls granddaughter cysts are developed. From the granular layer of the parent and

daughter cysts buds arise which develop into brood capsules. From the lining membrane the little outgrowths arise and gradually develop into bodies known as scolices, which represent in reality the head of the *tænia echinococcus* and present four sucking disks and a circle of hooklets. Each scolex is capable when transferred to the intestines of a dog of developing into an adult tape-worm. The difference between the ovum of an ordinary tape-worm, such as the *tænia solium*, and the *tænia echinococcus* is in this way very striking. In the former case the ovum develops into a single larva—the *cysticercus cellulosæ*—whereas the egg of the *tænia echinococcus* develops into a cyst which is capable of multiplying enormously and from the lining membrane of which millions of larval tape-worms develop. Ordinarily in man the development of the echinococcus takes place as above mentioned and by an endogenous form in which the secondary and tertiary cysts are contained within the primary; but in animals the formation may be different, as the buds from the primary cyst penetrate between the layers and develop externally, forming the *exogenous* variety. A third form is the multilocular echinococcus, in which from the primary cysts buds develop which are cut off completely and are surrounded by thick capsules of a connective tissue, which join together and ultimately form a hard mass represented by strands of connective tissue enclosing alveolar spaces about the size of peas or a little larger. In these spaces are found the remnants of the echinococcus cyst, occasionally the scolices or hooklets, but they are often sterile.

The fluid of the echinococcus cysts is clear and limpid, and has a specific gravity from 1·005 to 1·009. It does not contain albumen, but may contain traces of sugar. As a rule, the cysts, when not degenerated, contain the hydatid heads or scolices or the characteristic hooklets.

Changes in the Cyst.—It is not known definitely how long the echinococcus remains alive, but it probably lives many years—according to some authors as long as twenty years. The most common change is death and the gradual inspissation of the contents and conversion of the cyst into a mass containing putty-like or granular material which may be partially calcified. Remnants of the chitinous cyst wall or hooklets may be found. These obsolete hydatid cysts are not infrequently found in the liver. A more serious termination is rupture, which may take place into a serous sac, or perforation may take place externally, when the cysts are discharged, as into the bronchi or alimentary canal or urinary passages. More unfavorable are the instances in which rupture occurs into the bile-passages or into the inferior cava. Recovery may follow the rupture and discharge of the hydatids externally. Sudden death has been known to follow the rupture. A third and very serious mode of termination is suppuration, which may occur spontaneously or follow rupture and is found most frequently in the liver. Large abscesses may be formed which contain the hydatid membranes.

Geographical Distribution of the Echinococcus.—The disease prevails

Icelanders who have immigrated to this country have also carried the disease with them. Ferguson of Winnipeg has reported.

most extensively in those countries in which man is brought into close contact with the dog, particularly when, as in Australia, the dogs are used extensively for herding sheep, the animal in which the larval form of the *tænia echinococcus* is most frequently found. In Iceland the cases are very numerous. In Europe the disease is not uncommon. In this country it is extremely rare and a great majority of all cases are in foreigners. Up to July, 1891, I have been able to find in the literature (and in the museums) only 85 cases in the United States and Canada.*

Distribution in the Body.—Of the 1,862 cases comprised in the statistics of Davaine, Cobbold, Finsen, and Neisser, the parasites existed in the liver in 953, in the intestinal canal in 163, in the lung or pleura in 153, in the kidneys, bladder, and genitals in 186, in the brain and spinal canal in 127, bone 61, heart and blood-vessels 61, other organs 158.† Of the 85 cases in this country, the liver was the seat of the disease in 59. Of 50 consecutive cases treated by Mosler at the Greifswald clinic, 36 involved the liver, 10 the lungs, 3 the right kidney, and 1 the spleen.

Symptoms.—(*a*) *Hydatids of the Liver.*—Small cysts may cause no disturbance; large and growing cysts produce signs of tumor of the liver with great increase in the size of the organ. Naturally the physical signs depend much upon the situation of the growth. Near the anterior surface in the epigastric region the tumor may form a distinct prominence and have a tense, firm feeling, sometimes with fluctuation. A not infrequent situation is to the left of the suspensory ligament, forming a tumor which pushes up the heart and causes an extensive area of dulness in the lower sternal and left hypochrondriac regions. In the right lobe, if the tumor is on the posterior surface, the enlargement of the organ is chiefly upward into the pleura and the vertical area of dulness in the posterior axillary line is increased. Superficial cysts may give what is known as the hydatid fremitus. If the tumor is palpated lightly with the fingers of the left hand and percussed at the same time with those of the right there is felt a vibration or trembling movement which persists for a certain time. It is not always present, and it is doubtful whether it is peculiar to the hydatid tumors or is due, as Briançon held, to the collision of the daughter cysts. Very large cysts are accompanied by feelings of pressure or dragging in the hepatic region, sometimes actual pain. The general condition of the patient is at first good and the nutrition little, if at all, interfered with. Unless some of the accidents already referred to occur, the symptoms indeed may be trifling and due only to the pressure or weight of the tumor.

Suppuration of the cyst changes the clinical picture into one of pyæmia. There are rigors, sweats, more or less jaundice, and rapid loss of weight. Perforation may occur into the stomach, colon, pleura, bronchi, or exter-

* American Journal of the Medical Sciences, October, 1882. Since that date Alfred Mann has collected for me 24 cases in addition to the 61 there reported.

† Dictionnaire Encyclopédique des Sciences Médicales, tome 32, 1885.

nally, and in some instances recovery has taken place. Perforation into the pericardium and inferior vena cava is fatal. In the latter case the daughter cysts have been found in the heart, plugging the tricuspid orifice and the pulmonary artery. Perforation of the bile-passages causes intense jaundice, and may lead to suppurative cholangitis.

An interesting symptom connected with the rupture of hydatid cysts is the development of urticaria, which may also follow aspiration of the cysts and is probably due to the absorption of toxic materials contained in the fluid.

Diagnosis. — Cysts of moderate size may exist without producing symptoms. Large multiple echinococci may cause great enlargement with irregularity of the outline, and such a condition persisting for any time with retention of the health and strength suggests hydatid disease. An irregular, painless enlargement, particularly in the left lobe, or the presence of a large, smooth fluctuating tumor of the epigastric region is also very suggestive, and in this situation, when accessible to palpation, it gives a sensation of a smooth elastic growth and possibly also the hydatid tremor. When suppuration occurs the clinical picture is really that of abscess and only the existence of previous enlargement of the liver with good health would point to the fact that the suppuration was associated with hydatids. Syphilis may produce irregular enlargement without much disturbance in the health, sometimes also a very definite tumor in the epigastric region, but it is usually firm and not fluctuating. The clinical features may simulate cancer very closely. In a case which I reported the liver was greatly enlarged and there were many nodular tumors in the abdomen. The post-mortem showed enormous suppurating hydatid cysts in the left lobe of the liver which had perforated the stomach in two places and also the duodenum. The omentum, mesentery, and pelvis also contained numerous cysts. As a rule, the clinical course of the disease would suffice to separate it clearly from cancer. Dilatation of the gall-bladder and hydronephrosis have both been mistaken for hydatid disease. In the former the movable character of the tumor, its shape, and the mucoid character of the contents suffice for the diagnosis. In some instances of hydronephrosis only the exploratory puncture could distinguish between the conditions. More frequent is the mistake of confounding a hydatid cyst of the right lobe pushing up the pleura with pleural effusion of the right side. The heart may be dislocated, the liver depressed, and dulness, feeble breathing, and diminished fremitus are present in both conditions. Frerichs lays stress upon the different character of the line of dulness; in the echinococcus cyst the upper limit presents a curved line, the maximum of which is usually in the scapular region. Suppurative pleurisy may be caused by the perforation of the cyst. If adhesions result, the perforation takes place into the lung, and fragments of the cysts or small daughter cysts may be coughed up. For diagnostic purposes the exploratory puncture should be used. As

stated, the fluid is usually perfectly clear or slightly opalescent, the reaction is neutral, and the specific gravity varies from 1·005 to 1·009. It is non-albuminous, but contains chlorides and sometimes traces of sugar. Hooklets may be found either in the clear fluid or in the suppurating cysts. They are sometimes absent, however, as the cyst may be sterile.

(*b*) *Echinococcus of the Respiratory System.*—The larvæ may develop primarily in the pleura and attain a large size. The symptoms are at first those of compression of the lung and dislocation of the heart. The physical signs are those of fluid in the pleura and the condition could scarcely be distinguished from ordinary effusion. The line of dulness may be quite irregular. As in the echinococcus of the liver, the general condition of the patient may be excellent in spite of the existence of extensive disease. Pleurisy is rarely excited. The cysts may become inflamed and perforate the chest wall. In a case of D. F. Smith's, of Walkertown, Ontario, a girl, aged twenty, had a running sore in the eighth left intercostal space. This was freely opened, and in the pus which flowed out were a number of well-characterized echinococcus cysts of various size. The patient recovered.

Echinococci occur more frequently in the lung than in the pleura. If small, they may exist for some time without causing serious symptoms. In their growth they compress the lung and sooner or later lead to inflammatory processes, often to gangrene, and the formation of cavities which connect with the bronchi. Fragments of membrane or small cysts may be expectorated. Hæmorrhage is not infrequent. Perforation into the pleura with empyema is common. A majority of the cases are regarded during life as either phthisis or gangrene, and it is only the detection of the characteristic membranes or the hooklets which leads to the diagnosis. The condition is usually fatal; only a few cases have recovered. Of the 85 American cases, in six the cysts occurred in the lung or pleura.

(*c*) *Echinococcus of the Kidneys.*—In the collected statistics referred to above the genito-urinary system comes second as the seat of hydatid disease, though it is rare in comparison with the affection of the liver. Of the 85 American cases, there were only three in which the kidneys or bladder were involved. The kidney may be converted into an enormous cyst resembling hydronephrosis.

The diagnosis is only possible by puncture and examination of the fluid. The cyst may perforate into the pelvis of the kidney and portions of the membrane or cysts may be discharged with the urine, sometimes producing renal colic. I have reported a case in which for many months the patient passed at intervals numbers of small cysts with the urine. The general health was little if at all disturbed, except by the attacks of colic during the passage of the parasites.

(*d*) *Echinococcus of the Nervous System.*—In this country very few instances have occurred in the brain. One or two reports indicate clearly that the common cystic disease of the choroidal plexuses has been mis-

taken for hydatids. Davies Thomas, of Australia, has tabulated 97 cases, including some of the *cysticercus cellulosæ*. According to his statistics, the cyst is more common on the right than on the left side, and is most frequent in the cerebrum.

The symptoms are very indefinite, as a rule, being those of tumor. Persistent headache, convulsions, either limited or general, and gradually developing blindness have been prominent features in many cases.

Multilocular Echinococcus.—This form merits a brief separate description, as it differs so remarkably from the usual type of the disease. About one hundred instances are on record, the great majority of which have occurred in Bavaria and in Switzerland. Only one case has been reported in the United States.* The patient was a German, who had been in the country five years. For a year previous to his death he was out of health, jaundiced, and somewhat emaciated. A fluctuating tumor was found in the right lumbar and umbilical regions, apparently connected with the liver. This was opened, and death followed from hæmorrhage. About a fourth of the right lobe of the liver was occupied by an irregular cavity with rough, ragged walls, which in places were from one to two inches in thickness and enclosed irregular small cavities. The lamellated cuticula characteristic of the echinococcus cyst was found lining these cavities. In some instances the tumor bears a striking likeness to colloid cancer, as on section it presents a fibrous stroma with cavities containing gelatinous material. They are often sterile—that is, without the hydatid heads or larvæ. This form is almost exclusively confined to the liver, and the symptoms resemble more those of tumor or cirrhosis. The liver is, as a rule, enlarged and smooth, not irregular as in the ordinary echinococcus. Jaundice is a common symptom. The spleen is usually enlarged, there is progressive emaciation, and toward the close hæmorrhages are common.

Treatment of Echinococcus Disease.—Medicines are of no avail. Post-mortem reports show that in a considerable number of cases the parasite dies and the cyst becomes harmless. Operative measures should be resorted to when the cyst is large or troublesome. The simple aspiration of the contents has been successful in a large number of cases, and as it is not in any way dangerous, it may be tried before the more radical procedure of incision and evacuation of the cysts. Suppuration has occasionally followed the puncture. Injections into the sac should not be practised. With modern methods surgeons now open and evacuate the echinococcus cysts with great boldness, and the Australian records, which are the most numerous and important on this subject, show that recovery is the rule in a large proportion of the cases. Suppurative cysts in the liver should be treated as abscess. Naturally the outlook is less favorable. The practical treatment of hydatid disease has been greatly

* Delafield and Prudden, Pathological Anatomy, third edition, page 317.

The parasite is very rare in this country. Flint refers to a Missouri case in which from

W. H Evans of Sedalia Mo has recently had two cases in which the parasites were passed with the urine.

advanced by Australian surgeons. The recent work of James Graham, of Sydney, may be consulted for interesting details in diagnosis and treatment.

V. PARASITIC ARACHNIDA.

(1) **Pentastomes.**—(*a*) The *pentastomum tænioides* has a somewhat lancet-shaped body, the female from three to four inches in length, the male about an inch in length. The body is tapering and marked by numerous rings. The adult worm infests the frontal sinuses and nostrils of the dog, more rarely of the horse. The larval form, which is known as the *pentastomum denticulatum*, is found in the internal organs, particularly the liver, but has also been found in the kidney. The adult worm has been found in the nostril of man, but is very rare and seldom occasions any inconvenience. The larvæ are by no means uncommon, particularly in parts of Germany.

(*b*) The *pentastomum constrictum*, which is about the length of half an inch, with twenty-three rings on the abdomen, was found by Aitken in the liver and lungs of a soldier of a West Indian regiment.

~~The only case of pentastomes which, so far as I know, has been reported in this country is the one referred to in Flint's Practice of Medicine.~~ ~~From~~ 75 to 100 of the parasites were expectorated. The liver was enlarged and the parasites probably occupied this region. In 1869 I saw a specimen which had been passed in the urine by a patient of James H. Richardson, of Toronto.

(2) **Demodex (Acarus) Folliculorum.**—A minute parasite, from 0·3 millimetre to 0·4 millimetre in length, which lives in the sebaceous follicles, particularly of the face. It is doubtful whether it produces any symptoms. Possibly when in large numbers they may excite inflammation of the follicles, leading to acne.

(3) **Acarus** (or **Sarcoptes**) **Scabiei** (*Itch Insect*).—This is the most important of the arachnid parasites, as it produces troublesome and distressing skin eruptions. The male is ·23 millimetre in length, and ·19 millimetre in breadth; the female is 0·45 millimetre in length and 0·35 millimetre in width. The female can be seen readily with the naked eye and has a pearly-white color. It is not so common a parasite in the United States and Canada as in Europe.

The insect lives in a small burrow, about one centimetre in length, which it makes for itself in the epidermis. At the end of this burrow the female lives. The male is seldom found. The chief seat of the parasite is in the folds where the skin is most delicate, as in the web between the fingers and toes, the backs of the hands, the axilla, and the front of the abdomen. The head and face are rarely involved. The lesions which result from the presence of the itch insect are very numerous and result largely from the irritation of the scratching. The commonest is a papular

and vesicular rash or, in children, an ecthymatous eruption. The irritation and pustulation which follow the scratching may completely destroy the burrows, but in typical cases there is rarely any doubt as to the diagnosis.

The treatment is simple. It should consist of warm baths with a thorough use of a soft soap, after which the skin should be anointed with sulphur ointment, which in the case of children should be diluted. An ointment of naphthol (drachm to the ounce) is very efficacious.

(4) **Leptus Autumnalis** (*Harvest Bug*).—This reddish-colored parasite, about one half millimetre in size, is often found in large numbers in fields and in gardens. They attach themselves to animals and man with their sharp proboscides, and the hooklets of their legs produce a great deal of irritation. They are most frequently found on the legs. They are readily destroyed by sulphur ointment or corrosive-sublimate lotions.

Several varieties of ticks are occasionally found on man—the *Ixodes ricinus* and the *Ixodes americanus*, which are met with in horses and oxen.

VI. PARASITIC INSECTS.

(1) **Pediculi** (*Phthiriasis; Pediculosis*).—There are three varieties of the body louse, which are found only in persons of uncleanly habits.

Pediculus Capitis.—The male is from 1 to 1·5 millimetre in length and the female nearly 2 millimetres in length. The color varies somewhat with the different races of men. It is light gray with a black margin in the European, and very much darker in the negro and Chinese. They are oviparous, and the female lays about sixty eggs, which mature in a week. The ova are attached to the hairs, and can be readily seen as white specks, known popularly as nits. The symptoms are irritation and itching of the scalp. When numerous they may excite an eczema or a pustular dermatitis, which causes crusts and scabs, particularly at the back of the head. In the most extreme cases the hair becomes tangled in these crusts and matted together, forming at the occiput a firm mass which is known as *plica polonica*, as it was not infrequent among the Jewish inhabitants of Poland.

Pediculus Corporis (*Vestimentorum*).—This is considerably larger than the head louse. It lives on the clothing and in sucking the blood causes minute hæmorrhagic specks, which are very common about the neck, back, and abdomen. The irritation of the bites may cause urticaria, and the scratching is usually in linear lines. In long-standing cases, particularly in the old dissipated characters, the skin becomes rough and greatly pigmented, a condition which has been termed the vagabond's disease—*morbus errorum*—and which may be mistaken for the bronzing of Addison's disease.

Pediculus pubis differs somewhat from the other forms, and is found

in the parts of the body covered with short hairs, as the pubes; more rarely the axilla and eyebrows, and even the scalp.

The *taches bleuâtres* are stated by French writers to be excited by the irritation of pediculi. They are certainly associated with them in a considerable number of cases, but, if really caused by these parasites, it is difficult to understand why they should only be present with fever.

Treatment.—For the *pediculus capitis*, when the condition is very bad, the hair should be cut short, as it is very difficult to destroy thoroughly all the nits. Repeated saturations of the hair in coal oil or in turpentine are usually efficacious, or with lotions of carbolic acid, one to fifty. Scrupulous cleanliness and care are sufficient to prevent recurrence. In the case of the *pediculus corporis* the clothing should be placed for several hours in a disinfecting oven. To allay the itching a warm bath containing four or five ounces of bicarbonate of soda is useful. The skin may be rubbed with a lotion of carbolic acid, two drachms to the pint, with two ounces of glycerin. For the *pediculus pubis* white precipitate or ordinary mercurial ointment should be used, and the parts should be thoroughly washed two or three times a day with soft soap and water.

(2) **Cimex Lectuarius** (*Common Bed-bug*).—This parasite is from three to four millimetres in length and has a reddish-brown color. It lives in the crevices of the bedstead and in the cracks in the floor and in the walls. It is nocturnal in its habits. The peculiar odor of the insect is caused by the secretion of a special gland. The parasite possesses a long proboscis, with which it sucks the blood. Individuals differ remarkably in the reaction to the bite of this insect; some are not disturbed in the slightest by them, in others the irritation causes hyperæmia and often intense urticaria. Thorough fumigation with sulphur or scouring with corrosive-sublimate solution destroys them.

(3) **Pulex Irritans** (*The Common Flea*).—The male is from 2 to 2·5 millimetres in length, the female from 3 to 4 millimetres. The flea is a transient parasite on man. The bite causes a circular red spot of hyperæmia in the centre of which is a little speck where the boring apparatus has entered. The amount of irritation caused by the bite is variable. Many persons suffer intensely and a diffuse erythema or an irritable urticaria develops; others suffer no inconvenience whatever.

The *pulex penetrans* (*sand-flea; jigger*) is found in tropical countries, particularly in the West Indies and South America. It is much smaller than the common flea, and not only penetrates the skin, but burrows and produces an inflammation with pustular or vesicular swelling. It most frequently attacks the feet. It is readily removed with a needle. Where they exist in large numbers the essential oils are used on the feet as a preventive.

VII. PSEUDO-PARASITES (*Myiasis*).

Of these, the most important are the larvæ of certain diptera, particularly the flesh flies—*creophila*. The condition is called myiasis.

The most common form is that in which an external wound becomes *living*, as it is called. This myiasis vulnerum is caused by the larvæ of either the blue-bottle or the common flesh fly. The larvæ can be removed readily with the forceps; if there is any difficulty, thorough cleansing and the application of an antiseptic bandage is sufficient to kill them. The ova of these flies may be deposited in the nostrils, the ears, or the conjunctiva—the myiasis narium, aurium, conjunctivæ. This invasion rarely takes place unless these regions are the seat of disease. In the nose and in the ear the larvæ may cause serious inflammation.

The cutaneous myiasis may be caused by the larvæ of the *musca vomitoria*, but more commonly by the bot-flies of the ox and sheep, which occasionally attack man. This condition is rare in temperate climates. Matas has described a case in which œstrus larvæ were found in the gluteal region. In parts of Central America the eggs of another bot-fly, the *dermatobia*, are not infrequently deposited in the skin and produce a swelling very like the ordinary boil.

Myiasis interna may result from the swallowing of the larvæ of the common house fly or of species of the genus *anthomyia*. There are many cases on record in which the larvæ of the *musca domestica* have been discharged by vomiting. Instances in which dipterous larvæ have been passed in the fæces are less common. Finlayson, of Glasgow, has recently reported an interesting case in a physician, who, after protracted constipation and pain in the back and sides, passed large numbers of the larvæ of the flower fly—*anthomyia canicularis*. Among other forms of larvæ or *gentles*, as they are sometimes called, which have been found in the fæces are those of the common house fly, the blue-bottle fly, and the *techomyza fusca*. The larvæ of other insects are extremely rare. It is stated that the caterpillar of the tabby moth has been found in the fæces.

Here may be mentioned among the effects of insects the remarkable *urticaria epidemica*, which is caused in some districts by the procession caterpillars, particularly the species *cnethocampa*. There are districts in the Kahlberger Schweiz which have been rendered almost uninhabitable by the irritative skin eruptions caused by the presence of these insects, the action of which is not necessarily in consequence of actual contact with them.

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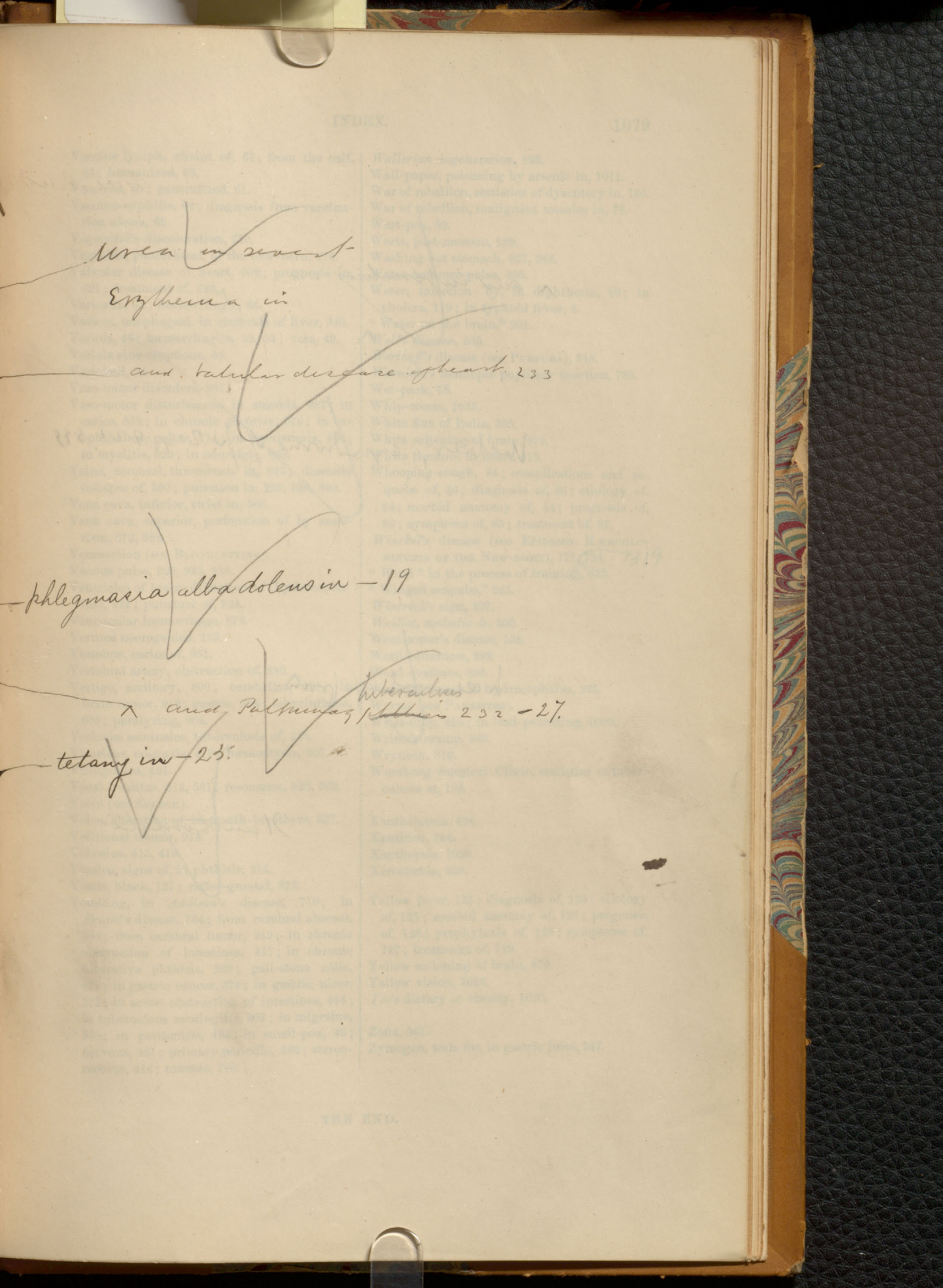
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